The Effectiveness of Fatigue-Monitoring In Professional Football

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Abstract

Player monitoring is common practice in elite sporting environments for practitioners to gain an advantageous insight into the general fatigue and recovery status of athletes and has previously been investigated. However, the application and sensitivity of fatigue markers for elite football players are yet to be fully established. The physical demands of football have changed over time, with increases in both physical and technical parameters observed in recent years (Barnes et al., 2014; Bush et al., 2015). Higher physical demands, coupled with periods of fixture congestion require players to compete in up to three games per-week, and has resulted in an increased need to monitor the associated fatigue response to avoid performance decrements and the inherent risk of injury (Nedelec et al., 2012; Zouhal et al., 2013).

Monitoring the impact of training and games on players' physiological and physical responses can allow practitioners to make informed decisions to ensure athletes are at peak physical performance prior to the next fixture through training modification and/or adjustment of individual player's recovery strategies. These markers of fatigue should be sensitive to both acute and chronic fluctuations in training/match load. Nevertheless, most research in elite football has been conducted over a short period in-season with limited observation of cumulative and chronic fatigue. Therefore, the primary aim of this thesis was to evaluate the sensitivity of conventional measures of fatigue (countermovement jump indices, creatine kinase concentrations and urine osmolality) on elite senior first team football players (now referred to as elite football players). The second aim was to assess whether these measures are sensitive to the stresses of an elite competitive football season.

The aim of the first study (Chapter 4) was to examine the acute changes from 90-minutes (min) of competitive match-play in countermovement jump (CMJ), creatine kinase (CK) and urine osmolality (Uosm) scores as part of routine testing and monitoring of elite football players over the course of a season. The results indicated that CMJ height derived from a 'Just Jump System' (Just Jump System, Perform Better Limited, Southam, Warwickshire, UK) could detect significant impairments at 48-hours (h) post-match in physical performance after performing 90-min of competitive match play (-3.9 %, P = 0.014, ES = 0.37). However, the question was raised whether the impairment was a meaningful change in performance and/or of clinical significance. There were marked increases in CK concentration, measured via capillary blood, 24-h pre to 48-h post-match (41 %, P = 0.005, ES = -0.91), however post-match concentrations were low in comparison to the large reference range reported in literature and would suggest minimal muscular or fatigue related stress was present. Urine osmolality measured using a handheld urinary refractometer (Osmocheck pocket pal OSMO, Vitech Scientific Ltd, Japan) displayed no significant change 24-h pre to 48-h post-match reflecting that elite football players are able to achieve rehydration by 48-h post-game, or the equipment employed within the limitations of the research environment was not sensitive to changes.

Chapter 5 mirrored that of Chapter 4 and used a more advanced assessment technique for physical performance via a force platform (HUR Labs Force Platform 3.8.0.2, Kokkola, Finland). Additional measures of physical performance (contraction time [CT], flight time [FT], the ratio between flight time and contraction time [FT:CT], peak power [PP], max force [MF], take-off velocity [TV], average power [AP], average force [AF]) were taken. This was due to research suggesting that concentric focused outputs from the 'push-off' phase of the jump, such as CMJ height, lacks the resolution to detect

neuromuscular fatigue (NF) in elite athletes. The findings from this study demonstrated CMJ FT and AP (concentric based outputs) to be most sensitive to fatigue 48-h post-match (-2.4 %, ES = -0.45 and -7.3 %, ES = -0.63, respectively) and moderately associated with external load parameters completed during 90-min of competitive match-play (r = -0.40 to -0.50). Decrements were also noted in FT:CT (-7.4 %, ES = -0.39) reflecting changes to concentric push-off (FT) and altered movement strategy (CT), but was not sensitive to match external load. The output measures of CMJ height and TV also demonstrated sensitivity to detect signs of NF but CT, PP and MF did not. Creatine kinase concentrations at 48-h post-match showed no association to match external load but were sensitive to the overall demands of 90-min of competitive football ($P = 0.001$, ES = 0.66). Similar to the findings of Chapter 4, Uosm did not reveal any significant difference at 48-h post-match ($P < 0.05$). The position specific changes in these markers were as expected, showing the positon of central midfielder to display the greatest changes in CK and CMJ outputs likely attributable to their position covering the greatest total distance, high intensity running distance and number of high intensity actions. The studies comprising Chapter 4 and 5 show that CMJ testing and CK concentrations display particular promise as acute, simple, non-invasive assessments for monitoring the recovery-fatigue status of elite football players' 48-h post-match.

The aim of the third study (Chapter 6) was to examine the longitudinal fluctuations in the physiological and physical performance markers from Chapter 4 and 5 across a competitive season. To the author's knowledge, this was the first study to address weekly variations in these measures over a competitive season. The results demonstrated large increases in CK concentration but minimal fluctuation over a competitive season [62 to 159 %, $P < 0.05$], and indicated that CMJ MF can be increased (5.1 to 7.0 %, $P < 0.05$) despite fluctuations in movement strategy focused outputs (TV, FT:CT and AP) possibly

indicating low-level NF. Therefore, the results from this chapter support the idea that skilled performers' are thought to exhibit greater movement variability to help achieve consistent performance outcomes. Hydration status measured via Uosm demonstrated significant increases to baseline over the season (30 to 84 %, $P < 0.05$), although the findings indicated that players were able to maintain a euhydrated state that would be unlikely to negatively affect performance output. Chapter 6 additionally provided 95 % reference ranges and normative values for the recovery-fatigue variables in this sub-group of elite football players to aid the interpretation of clinically meaningful changes from baseline.

The aim of the last experimental Chapter (Chapter 7) was to observe the association between markers of training/match load and the conventional markers of recovery-fatigue over a competitive season, to determine the sensitivity of these markers to preceding and cumulative load. The high inter-individual variability in the recovery-fatigue variables was addressed by transforming raw scores into 'Z-scores' for the analysis. The results from regression analysis showed that the models used explained low proportions of variance in the physiological and physical performance parameters used (6 to 18 %). Creatine kinase had a weak positive association to cumulative match-time over the season and Uosm to the time-trend variable 'match-week' (week number of the season; $P < 0.05$). Match-week also demonstrated significant associations to increases in CMJ AF. Further, the results displayed weak, marginally significant, associations between CMJ CT and FT:CT to preceding match-load. All recovery-fatigue variables used demonstrated poor sensitivity to training load outputs measured via GPS over a season. The findings suggest that elite football players, who play regularly, demonstrate the capability to maintain physical output and be sufficiently recover by 24-h pre-match (for a Saturday fixture) despite a congested match schedule.

The overall findings of this thesis have shown that simple output measures from a CMJ are able to detect impairments in physical performance and signs of NF 48-h post competitive match-play in elite football players. Further, concentric derived output measures from the 'push-off' phase previously criticised in research have displayed sensitivity to match external load. Across a competitive season, players are able to maintain MF and PP outputs despite signs of low-level NF via changes in 'jump-strategy' (e.g. TV, FT:CT and AP). Creatine kinase, although sensitive to overall match-play, displays changes of minimal clinical significance 48-h post-match. However, CK did demonstrate sensitivity to cumulative match-min played across the season. Therefore, if interpreted correctly CK concentrations may provide practitioners with insights into cumulative fatigue/muscular stress. Incorporating additional biomarkers may further enhance detection of physiological and physical stress related changes to match and training demands. Lastly, the monitoring of hydration status via Uosm reflects that players are able to maintain a state of euhydration across a season but shows no associations to preceding match or training demands, perhaps a limitation of the equipment employed in the research environment was not sensitive to changes. Therefore, monitoring for spikes in CK concentrations and impairments in CMJ performance, when interpreted correctly, may provide clinically relevant insights of individual players prior to the next training session and/or fixture, ultimately equipping practitioners with an advantageous knowledge concerning recovery-fatigue status.

Key Words: soccer; creatine kinase; countermovement jump; hydration; performance monitoring; neuromuscular fatigue

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Statement of Original Authorship

The work contained in this thesis has not been previously submitted for a degree or diploma at any other higher education institutions to the best of my knowledge and belief. This thesis is structured based on the regulations and procedures governing the award of the degree of 'Doctor of Philosophy by Thesis via MPhil' by University of Bolton, UK**.**

C. E. Beattie

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List of Abbreviations

ADP	Adenosine diphosphate
AF	Average force
AP	Average power
ARF	Australian rule football
ATP	Adenosine triphosphate
BAS	Baseline
BUN	Blood urea nitrogen
C	Cortisol
Ca2+	Calcium
CHO	Carbohydrate
CI	Confidence interval
CK	Creatine kinase
cm	Centimetres
CMJ	Counter-movement jump
CNS	Central nervous system
CT	Contraction time
CV	Coefficient of variation
DHA	Docosahexaenoic acid
DHEA	Dehydroepiandrosterone
DOMS	Delayed onset of muscle soreness
DVs	Dependent variables
EIMD	Exercise induced muscle damage
EPA	Eicosapentaenoic acid
ES	Effect size
EXS	Explosive sprints
FFA	Free fatty acid
FFM	Fitness fatigue model
FL	Fluid intake
FT	Flight time
FT:CT	Flight Time: Contraction Time
GAS	General adaption syndrome
GH	Growth hormone
GPS	Global positing system
GTO	Golgi tendon organs
h	Hour
H.I Acc	High intensity accelerations (GPS)
H.I Dec	High intensity decelerations (GPS)
H+	Hydrogen
HAcc	Number of high accelerations (Prozone®)
HDec	Number of high decelerations (Prozone®)
HIN	High intensity number
HIR	High intensity running
HSR	High speed running
IL-6	Interleukin-6
IQR	Interquartile ranges
IVs	Independent variables
JJS	Just-jump system

K+	Potassium
Kg	Kilogram
km	Kilometre
L	Litre
LOA	Limits of agreement
m	Metres
M.I Acc	Moderate intensity accelerations (GPS)
M.I Dec	Moderate intensity decelerations (GPS)
MD +2	Match day plus 2
MD -1	Match day minus 1
MAcc	Number of medium accelerations (Prozone®)
MDec	Number of medium decelerations (Prozone®)
MF	Max force
Min	Minutes
ml	Millilitre
MR	Mouth rinse
ms	Milliseconds
MVC	Maximum voluntary contraction
N	Newtons
Na	Sodium
NF	Neuromuscular fatigue
NFL	No fluid
O2	Oxygen
PCr	Phosphocreatine
pH	Power of hydrogen
Pi	Inorganic phosphate
PL	Player load
PP	Peak power
RFD	Rate of force development
RR	Relative risk
s	Seconds
SD	Standard deviation
SEM	Standard error of measurements
SR	Sarcoplasmic reticulum
SSC	Stretch shortening cycle
SWC	Smallest worthwhile change
T	Testosterone
TD	Total distance
THIR	Total high intensity running
TSD	Total sprint distance
TV	Take-off velocity
u/l	Units per litre
Uosm	Urine osmolality
YYIRT	Yo-Yo intermittent recovery test

Chapter 1: General introduction

1.1: Background

The success in elite football is determined by slight margins, with differences between success and failure quantified by the smallest of increments (Drust et al., 2005). The decisions made by a team or an individual can lead to events that ultimately result in a defeat or a victory (Casanova et al., 2013). For example, the 2014 UEFA Champions League Final between Real Madrid and Atlético Madrid, where Sergio Ramos equalised for Real Madrid in the third minute of added time to send the game to extra time, after which Real Madrid went on to win is a prime example of this. This further highlights the ever-increasing importance of small margins and athletic development in elite football. There is a need for the development and understanding of scientific processes to aid players, managers and sport scientists to analyse performance in football (Drust et al., 2007; Bourdon et al., 2017). Over recent years elite football has become more physically demanding (Barnes et al., 2014; Wallace and Norton, 2014). Fixture congestion is of contemporary concern, requiring players to compete in up to three games per-week and 60 to 70 matches over a full season, with as little as 72-hours (h) recovery in-between matches (Barnes et al., 2014; Carling et al., 2015; Abaïda and Dupont, 2018). The repetition of matches can lead to a chronic fatigue among the players who play regularly as the recovery time between such intense competition periods may be insufficient to restore physiological homeostasis (Fatouros et al., 2010; Abaïda and Dupont, 2018).

Elite football is an intermittent sport in which the aerobic energy system is highly taxed (Bangsbo et al., 2006), football matches have shown to elicit over 70 % of maximum oxygen consumption, emphasising the need of a high aerobic capacity (Reilly, 1997; Bangsbo et al., 2006). Although 30 to 60 % of the time during a match is spent at a low intensity, such as walking, jogging, or low/moderate speed running, the players are required to perform between 150 to 250 brief intense actions (Mohr et al., 2003). Thus,

the stochastic nature of football requires a high level of anaerobic energy turnover at certain times (Bangsbo, 1994; Bangsbo et al., 2006). The subsequent stress associated with these high-energy demands often temporarily impairs a player's performance (Ispirlidis et al., 2008; Fatouros et al., 2010). Sport performance and the outcome of competitive team games depends highly on the ability of individual players to sustain high levels of physical, technical and psychological skills throughout competition (Knicker et al., 2011; Zouhal et al., 2013). Fatigue can result in the inability to sustain such factors and can adversely influence footballing performance and the inherent risk of injury (Nedelec et al., 2012; Zouhal et al., 2013). Fatigue is a multifaceted, complex phenomenon, making it difficult to define and measure. However, match-related fatigue can be described as a decline in physical performance during the hours and days post-match, resulting in players performing at sub-optimal levels of physiological function (Rampinini et al., 2011). It is likely that players may suffer from transient, cumulative, and residual fatigue following training and competition (Mohr et al., 2003; Knicker et al., 2011). Acute and residual fatigue presents itself by a decline in physiological status and physical performance over the following hours and days post competition (Ispirlidis et al., 2008). Previous studies show that some components of physical performance and physiological status may still be impaired 72-h following match-play in comparison to pre-match baselines (Mohr et al., 2003; Andersson et al., 2008; Ispirlidis et al., 2008; Fatouros et al., 2010). Nonetheless, there is limited understanding on how best to quantify longer lasting neuromuscular fatigue (NF) within an elite sporting environment.

To manage the high physical demands of training and match-play as well as scheduling recovery, monitoring training load plays a crucial role inducing sport specific adaptation and avoiding the negative sequel of excessive fatigue (Impellizzeri et al., 2004; Ekstrand et al., 2013). While designing structured training programmes appears to be the first step

in performance management, the second most important step towards maximising an adaptive response is monitoring the impact of training and games on players' physiological responses. The use of player monitoring tools in elite sports is becoming increasingly popular for practitioners to gain an advantageous insight into the general fatigue and recovery status of athletes. In order to make informed decisions on player status it is important that tests are reliable and sensitive enough to detect meaningful changes in performance (Gathercole et al., 2015). There are standardised laboratory methods for the assessment of NF, however they are also invasive, time consuming and costly (Tofari et al., 2016). Attention in the literature has been focused upon developing valid, reliable and non-invasive monitoring tools to provide an indication of physiological fatigue status in athletes (Meeusen et al., 2013; Halson, 2014). Testing in-field during a day-to-day training environment of high-performance sports may include vertical jump testing, hydration testing, hormone markers, and wellness questionnaires (Halson 2014; Djaoui et al., 2017). Methods such as these are preferred due to their convenience, greater ecological validity and easy implementation to allow regular assessment of large groups. However, such tests/markers should be sensitive to preceding exercise load if it is a valid measure of fatigue in the sporting context, yet this concept has received limited attention in the literature (Buchheit et al., 2013; Gastin et al., 2013; Thorpe et al., 2015; de Hoyo et al., 2016).

The most common in-field test for the assessment of NF is the counter-movement jump (CMJ) test (Gathercole et al., 2015). The CMJ test is widely used throughout sport due to its ease of use and low physiological strain on the athlete. The CMJ test demonstrates excellent day-to-day reliability, with reported coefficients of variations (CV) of 1 to 5 % (Cormack et al., 2008a; Slinde et al., 2008; Kenny et al., 2012), with CV values below 5 % considered reliable in a sporting population in order to determine whether an

observed change is of practical significance (Pyne, 2003; Buchheit et al., 2011). Hydration testing has also become a popular monitoring tool with research highlighting the adverse effects of dehydration on aerobic performance, anaerobic performance, and cognitive performance (Cheuvront et al., 2003). Urine osmolality (Uosm) is a cheap, practical measure of hydration status and although not the "gold standard", it can be used to assess and distinguish euhydration and dehydration if the first void in the morning is used (Cheuvront and Sawka, 2005). Another popular measure for monitoring fatigue is that of creatine kinase (CK) concentrations. Increased levels of plasma CK activity is generally accepted as an indicator of exercise-induced muscle damage (EIMD; Fielding et al., 1993; Hody et al., 2013). The collection of plasma CK is minimally invasive and can provide immediate feedback making its use advantageous in the sporting environment. Due to the multifaceted nature of fatigue, a single marker alone cannot provide enough diagnostic information on the physical and physiological status of elite football players. Therefore, several markers in combination (CMJ, Uosm and CK) may provide practitioners with a more holistic understanding of fatigue on physical and physiological function.

Research in elite sport has addressed neuromuscular and endocrine responses to training and competition (Edwards et al., 2007; Cormack et al., 2008b; Cormack et al., 2008c; Owen et al., 2013; Thorpe et al., 2015; de Hoyo et al., 2016), but only a small amount of research has investigated season-long responses (Cormack et al., 2008c; Faude et al., 2011; Rowell et al., 2018, Nowakowska et al., 2019). As match frequency fluctuates during the season there is uncertainty regarding the expected pattern of response in these variables, and their usefulness in determining the impact of training and competition on changes in physical performance and physiological status of elite players. Fatigue can manifest over time, perhaps when the body has not had time to adapt to exercise stress

(Selye, 1950; Kentta and Hassmen, 1998). Therefore, a valid marker of fatigue should be sensitive to both acute and chronic fluctuations in training and match load (Meeusen et al., 2013). There is an abundance of literature that has examined the sensitivity of potential fatigue measures to daily and weekly fluctuations in training load in elite team sport athletes (Buchheit et al., 2013; Gastin et al., 2013; Thorpe et al., 2015). However, observing fatigue markers over such a relevantly short period will only allow for the observation of acute and short-term debilitating effects of exercise-induced fatigue. More research is needed to expose the longitudinal fluctuations to the stresses of an elite football season.

Justification for monitoring player fatigue in a professional club setting is largely founded on scientific literature from study populations of non-elite, academy or reserve squad players. The genetic makeup and training history of professional, senior athletes may vary to that of athletes at a lower level or non-athletes (Burgess, 2017). Therefore, they may respond differently to training stress and recovery over time (Barnett, 2006). Training history, age and match exposure are all factors that may alter the morphological make-up of athletes. Thus, findings from studies of differing populations may not be applicable to elite senior football players. Further, studies in general tend to investigate fatigue responses following a single match (Ispirilidis et al., 2008; Nedelec et al., 2014; de Hoyo et al., 2016). The 'one-off' data collected would not account for the recognised large match-to-match variation in physical demands (Carling et al., 2016) and can lead to an inaccurate interpretation of a players' physiological response. One of the first investigations to examine the effects of accumulated load on fatigue indices in elite senior football players was conducted by Thorpe et al. (2017). However, their research was conducted over a 17-day in-season competitive period and accumulated fatigue was only examined over a four-day period. Fatigue can manifest over a longer period of exposure

than a short block of training/competition. Chronic fatigue may have long-term debilitating effects such as performance decrements and an increased injury risk (Ekstrund et al., 2013). Creatine kinase concentrations over a professional football season have been researched more extensively than CMJ (Coelho et al., 2011; Scott et al., 2016). Scott et al. (2016) examined the relationship between physical match performance and CK concentration 48-h post-match across a season in the English Premier League. Their results demonstrated a marked difference in CK concentration pre- to post-match, but they were unable to identify any significant correlations between physical match performance and CK concentration. Recently, in elite Polish football players, Nowakowska et al. (2019) demonstrated a weak positive correlation between CK concentration and cumulative match-time in midfielders/defenders over a competitive season. However, the time-point of data collection at 24-h post-match is commonly a day focused on recovery in weekly micro-cycles and would therefore not influence training load modification for those players who have played a significant amount of match-mins. Further, de Hoyo et al. (2016) analysed both CK and CMJ variables post one competitive match, however the 'one-off' data collection and the cohort used of under 19-year-old elite footballers limits the studies ability to generalise the findings to that of senior professional footballers. While a large body of research proliferates in football, the ecological validity of investigations should be questioned on their applicability to elite, senior level, football players due to the populations and protocols used. There is a need for the observation of fatigue responses in specific sub-groups of elite football players regularly undertaking competitive match-play over the course of a professional season.

1.2: Statement of the problem

In the first instance, the research studies compromising this thesis are concerned with evaluating the sensitivity of conventional markers of fatigue used in elite, senior level, football players. As previously mentioned, although research is plentiful in elite sport on the acute changes of fatigue markers, the applicability of these findings to elite, senior level, football is somewhat limited due to the dissimilar physiological demands to that of other team sports such as Rugby Union, Rugby League, Australian Rules football and American football. Therefore, it is important to determine which potential markers are most sensitive to changes in load associated with specific sports and athletic sub-groups. This body of work builds on previous research by expanding the analysis to focus solely on elite first team, senior level, football players' regularly undertaking 90-min of competitive match-play over a season. In addition, this thesis is concerned with examining season-long responses to the accumulation of fatigue that may manifest due to fixture congestion and limited player rotation. This thesis will investigate whether these commonly used markers of fatigue can be relied upon and are sensitive enough to indicate when a player is fatigued to an extent that might affect their performance.

The findings from the research studies undertaken during the doctoral studies have the potential to provide practitioners in elite football with the most applicable objective marker of fatigue to use with elite first team players. Such objective markers, sensitive to training and match load, may assist the decision-making capabilities of practitioners and/or coaches to ensure athletes are at peak physical performance prior to the next fixture through training modification and/or adjusting individual player's recovery strategies.

1.2.1: Aims of the Thesis

To investigate the effectiveness and sensitivity of commonly collected fatigue markers in professional, senior level, football players in detecting meaningful changes in physical performance and physiological status.

Summary of the aims of this thesis are:

1. To assess the acute changes in physiological and physical performance markers, from multiple games across a season, after completing 90-min of competitive football in elite players 24-h pre-match to 48-h post-match (Chapter 4).

2. To analyse the sensitivity of physiological and physical performance markers to preceding match-load from multiple competitive games across a season, in elite players (Chapter 5).

3. To examine the longitudinal fluctuations in physiological and physical performance markers across a season in elite players (Chapter 6).

4. To examine the association between markers of training/match load and physiological and physical performance markers over a football season to determine the sensitivity of these markers to preceding and cumulative load (Chapter 7).

1.2.2: Objectives

Realisation of the aims will be achieved through several objectives listed below:

1. **Aim 1: Objective:** To analyse the acute changes in commonly employed markers of fatigue (CMJ height, CK, urine osmolality) 24-h pre-match to 48-h post-match after completing 90-min of competitive football, and determine if these markers are able to detect clinically significant changes in physiological and physical performance when used on elite, senior level, football players. This will be achieved through study 1 (Chapter 4).

2. **Aim 2: Objective:** To analyse the sensitivity of CMJ force platform outputs, CK concentrations and urine osmolality to 90-min of competitive match-play and their association to physical match performance measures collected via Prozone® semi-automated multi-camera recognition system. This will be achieved through study 2 (Chapter 5).

3. **Aim 3: Objective:** To examine the weekly changes, in the fatigue markers used from Chapter 4a and 4b, across a season in comparison to a pre-season training baseline in elite, senior level, football players. Further, to determine the longitudinal fluctuations in these markers to potential signs of cumulative fatigue/non-functional overreaching. This will be achieved through study 3 (Chapter 6).

4. **Aim 4: Objective:** Examine associations between preceding and cumulative training/match load to the fatigue markers used in Chapters 4 and 5, from data collected over an entire competitive season. Further, to control for the high inter-individual variability, the fatigue markers will be reported relative to an individual player's absolute average and normal variation from the duration of

the season by reporting them as Z-scores. This will be achieved through study 4 (Chapter 7).

Chapter 2: Review of the Literature

2.1: Structure of the Review

The purpose of the literature review is to outline the scope of literature examining the physical demands and stresses in elite football followed by the definition of fatigue and its abundant use in scientific literature. It is not the intention to be a comprehensive review of fatigue in football or the role of fatigue in inducing training adaptations, but rather a review to familiarise the reader with the common markers of fatigue used in football. It is also the aim to bring together the relevant aspects of fatigue and review current systems for monitoring physiological fatigue, with methodological considerations for each method evaluated in reference to the regular use for monitoring in a high performance-training environment.

2.2: Physical Demands of Professional Football

Football players can be classed according to payment as elite (paid professional players), sub-elite (receive moderate remuneration to play football, but also rely on additional employment to generate income), or non-elite (amateur players) players. It is well known that football is characterised by low- (standing and walking) and high-intensity (running and sprinting) activities (Bloomfield et al., 2007). Further, it has been characterised as a physiologically demanding intermittent sport, taxing both the aerobic and anaerobic energy systems (Rampinini et al., 2011).

Elite football players cover a total of between 9 to 14 km distance during competitive match-play (Bradley et al., 2009; Di Salvo et al., 2009; Bradley et al., 2013; Andrzejewski et al., 2018), and over 18 % of this distance covered is attained while accelerating or decelerating (Akenhead et al., 2013). Players perform at an average intensity that is close to the anaerobic lactate threshold between 70 to 80 % of maximal oxygen uptake (Helgerud et al., 2001; Chamari et al., 2004). The demands of various playing positions in elite football players have been shown to differ greatly (Bangsbo, 1994; Abbott et al., 2018) – with central and wide midfielders covering the greatest total distance and wide midfielders and full backs covering the most high-intensity running (HIR: Bradley et al., 2009; Abbott et al., 2018). As such, the squad formation and tactical role of players could also dictate the amount of HIR undertaken during a match (Bradley and Noakes, 2013). Evidently, football is a multifaceted sport in which physical, tactical and technical factors amalgamate to influence performance. It is important to understand the myriad of factors that influence physical performance in football such as environmental factors, tactical system employed, home advantage, score-line, importance of game, substitutions and previous results in order to contextualise physical

performance outputs during 90-min of competitive football. (Bradley et al., 2009; Bradley and Ade, 2018, Bradley et al., 2020).

A difference in physical demands has also been observed depending on the tier of elite English football (Di Salvo et al., 2009; Gregson et al., 2010; Bradley et al., 2013). Bradley et al. (2013) found that players in the second and third divisions covered more HIR metres (m) than those in the Premier League (803, 881 vs 681 m, respectively), similar results were also found for total distance covered by sprinting (308, 360 vs 248 m, respectively). Similarly, Di Salvo et al. (2009) found that Championship players performed more HIR and sprint metres over those players in the Premier League.

Players are exposed to a high-volume of high-intensity manoeuvres during matches such as accelerations, decelerations, repeated sprints, tackles, jumps and duel plays with only short periods of recovery (Mohr et al., 2003). This high-volume and high-stress participation has the potential to cause transient, cumulative and residual fatigue (Mohr et al., 2003; Knicker et al., 2011). Fatigue may result in the inability to repeatedly cover distances during critical match-play situations and reduce technical capability that can in turn impact team performance and match outcome (Kellis et al., 2006; Reilly et al., 2008; Rampinini et al., 2011; Smith et al., 2016). High intensity bouts seen in football utilise anaerobic metabolism, but typically the duration of active play in football is 90-min, indicating that the primary energy source during the game is supplied via aerobic glycolysis (Da Silva et al., 2008; Alghannam, 2012; Zouhal et al., 2013). Sustaining these high-intensity actions throughout match-play is critical to be first to the ball, to create and move into space and generate goal-scoring opportunities (Carling et al., 2015).

Elite football has become more demanding over the years (Barnes et al., 2014; Wallace and Norton, 2014), with research demonstrating an increase of 30 to 35 % in HIR and sprint distance (SD) between 2006-07 and 2012-13 in the English Premier League (Barnes et al., 2014). Authors have suggested that the physical, technical and tactical demands of football are increasing together with positional roles and playing formations evolving (Carling et al., 2008; Bradley et al., 2011). Barnes et al. (2014) also noted increases in absolute number of both explosive and "leading" sprints over a shorter distance and an increase in maximal running speed, suggesting that the acceleration capability of players has developed. From the 2006-7 to 2012-13 season, technical factors also showed a vast increase in the number of passes (~40 %), a greater percentage of successful passes (~8 %), and an increased number of short and medium passes (6 vs. 9 and 13 vs. 20, respectively). These findings demonstrate a greater intensity of football match-play and a requirement for players to be physically conditioned to cope with these higher demands.

Alongside an increase in the physical demands of football, players must additionally cope with a higher number of competitive matches over a nine-month season, a congested game-schedule and lower squad numbers. Certain players may perform up to 70 matches per season, with up to three matches in seven days (Barnes et al., 2014; Abaïda and Dupont, 2018), taking into account club and international football commitments. For teams with lower squad numbers, select players will be required to perform repeatedly with minimal recovery time. This repetitive match-play can lead to chronic fatigue and an increased injury risk (Ekstrand et al., 2013). Research linking recovery time, injury risk and team performance has shown inconsistent findings (Doupont et al., 2010; Carling et al., 2012; Dellal et al., 2015), and is limited in its generalisability due to small sample sizes. Ekstrand et al. (2013) were one of the first to study a large sample size

using professional football teams, and their results demonstrated an increased injury risk in league matches with four or less days' recovery when compared with six or more days' recovery (Relative Risk [RR] 1.09, 95 % CI 1.00 to 1.18, and RR 1.32, 95 % CI 1.15 to 1.51, respectively). The hamstring and quadriceps muscle group were of particular risk with short recovery between matches. No associations were found between team performance and recovery (Ekstrand et al., 2013). However, a longitudinal study conducted by Hägglund et al. (2013) found that the league ranking of a football team is highly influenced by the injury status of fellow players. Football teams that can avoid injuries and keep their players on the pitch are more successful and win more matches. Knowledge of such findings highlights the importance of athletic monitoring and injury prevention in the overall success of a football team.

Notably, professional football players have high physiological demands placed upon them from both training and competition. In addition to this, the variance in actions performed during competitive team sport match-play between playing positions and individuals may result in different levels of fatigue experienced post-game (Manzi et al., 2010). Understanding these demands in professional football provides an insight into the possible fatigue mechanisms involved.

2.3: Fatigue

The term 'Fatigue' is used abundantly throughout literature, but it should be noted that behind this simple term lies an undoubtedly complex phenomenon with multi-faceted contributors (Halson, 2014). An early definition by Edwards (1983) states that fatigue is a:

"Failure to maintain the required or expected force (or power output)."

The word 'fatigue' originated from the Latin word *fatigare*, which originally meant: "to cause to break down" and later "to tire". In scientific literature, fatigue is used in a variety of contexts and the meaning of the word has been manipulated in order to best suit individual disciplines. Abbiss and Laursen (2007) stated the following diverse examples of fatigue depending on the discipline and scientific investigation being studied:

- Biomechanics: a decrement in force output of a muscle, or a reduction in efficiency.
- Psychology: the sensation or perception of tiredness, or a decrease in cognitive function.
- Physiology: a limitation of a specific physiological system, such as the inability of the heart to supply sufficient blood flow to working tissues or the failure in the muscle excitation-contraction coupling process.
- Neurology: reduced motor drive or neural activation.

Exercise-induced fatigue has long been of interest to sports scientists yet remains only partially understood (Shei and Mickleborough, 2013). Exercise fatigue has been defined as a physical activity induced impairment in performance (Knicker et al., 2011). It can be represented by the reduced capacity to generate the required work-rate and level of force where performance must be sustained for a prolonged period (Reilly et al., 2008; Bishop, 2012). In team sports such as football, work-rate is influenced by the demands of the competition on the entire team as a unit, and not only by self-chosen exercise intensity (Reilly et al., 2008). Several mechanisms have been identified that contribute to the decline seen in physical performance, however the exact aetiologies are yet to be

established (Fitts, 1994). These mechanisms may differ depending on the circumstance and the activity in which it is performed (Allen et al., 2008). Fatigue can be influenced by multiple factors such as the type of contraction (isometric, isotonic, and intermittent or continual), the type of stimulus (voluntary or electrical induced), the frequency, the duration and the intensity of exercise (Sahlin, 1992). Furthermore, the morphological, physiological and training status of the athlete may influence fatigue (Halson, 2014). These factors highlight the multifactorial nature of fatigue and consequently the inherent complexities of trying to monitor aspects of fatigue in elite athletes. Researchers have typically measured the effects of fatigue, for example a reduction in force or a sensation of tiredness, in an endeavour to understand the possible mechanisms behind it (Abiss and Laursen, 2007).

Throughout the remainder of this thesis, fatigue is discussed in the context of a reduction in overall performance capacity.

2.3.1: Central and Peripheral Fatigue

Exercise fatigue is said to comprise both central and peripheral aspects, when muscle function is impaired directly due to physiological processes that originate in the muscle and this phenomenon is called peripheral fatigue (Knicker et al., 2011). Muscular fatigue may arise not only due to peripheral changes that occur at or are distal to the neuromuscular junction, but also when the central nervous system (CNS) fails to drive the motor neurons adequately, highlighting the role of the nervous system in the maintenance of muscle performance (Fernandez-del-Olmo et al., 2013). The processes involved in the spinal cord and above are defined as central fatigue (Allen et al., 2008). Fatigue has the potential to arise at several points during the pathway between the CNS

and the contracting muscle (Allen et al., 2008). It therefore seems essential to recognise the most important contributors of fatigue, to ensure the most appropriate method is chosen to reduce the effects of fatigue and increase the preparedness of athletes.

Enoka (2002) outlined nine processes within the neuromuscular system, both central and peripheral factors that can be impaired during exercise (Figure 1). These include: (1) the activation of the primary motor cortex; (2) the central nervous system drive to the motor neurons; (3) the muscles or motor units that are activated; (4) neuromuscular propagation; (5) excitation-contraction coupling; (6) the availability of metabolic substrates; (7) the intracellular milieu; (8) the contractile apparatus and (9) muscle blood flow (Figure 2.1).

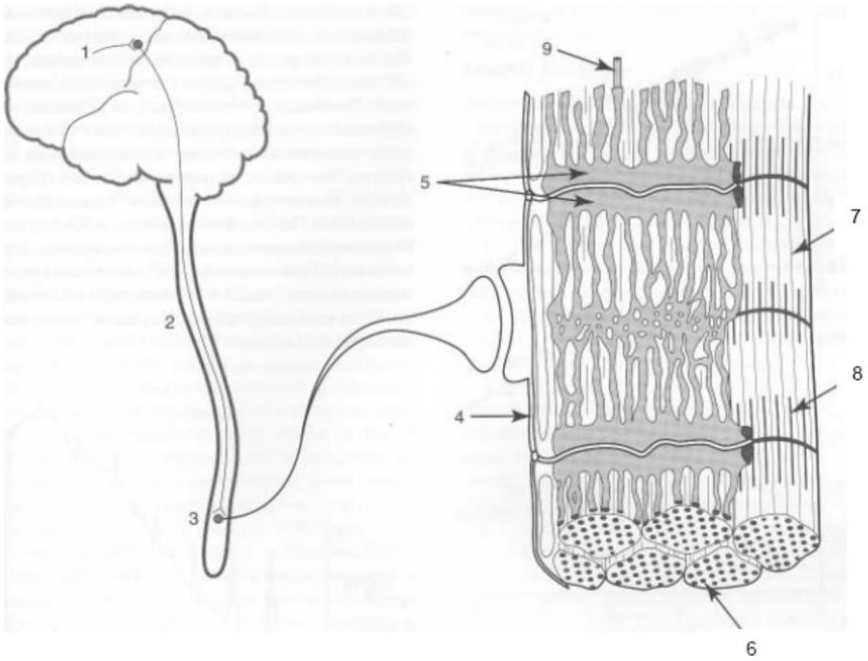


Figure 2.1. Locations of the nine processes that may contribute to fatigue during physical activity (Enoka, 2002; p.375).

Central fatigue has been suggested to be the main contributor related to drops in maximum voluntary contractions (MVC) immediately post-exercise (Rampinini et al., 2011). The motor cortex is a region of the brain that generates the neural impulses to travel along the CNS, to the motor neurons and to the muscle fibres, that begins the process of muscle contraction. Research has demonstrated, using involuntary electrical stimulation, that following maximal contractions the reductions in force were partly explained by a reduced output from the motor cortex (Gandevia et al., 1996). Additionally, by stimulating the motor cortex directly research has shown that maximal torque of the elbow flexors could be increased (Taylor et al., 2000). Such research helps to explain how sub-optimal output from the motor cortex can be a contributing factor to reduced voluntary muscle activation, and a component of central fatigue.

A motor unit consists of a motor neuron and the muscle fibres it innervates. Motor unit recruitment can also be altered by sensory receptors, such as from Golgi tendon organs (GTOs). These receptors play an important role in central fatigue models to preserve the overall integrity of the system through mechanisms such as motor unit decruitment (Allen et al., 2008). It is likely that these fatigue mechanisms can prevent the level of excess muscle contraction that could result in failure or damage (Allen et al., 2008). This follows the 'central governor' theory, which contends that the body functions as a complex system during exercise. The afferent feedback from multiple central and peripheral sensors enables the athlete to subconsciously 'anticipate' the demands of the exercise task, and select the best pacing strategy to accomplish it without any loss of cellular homeostasis through a reduction in efferent neural commands (Gibson and Noakes, 2004; Noakes et al., 2005; Lambert et al., 2005). Whilst many researchers make a distinction between central and peripheral fatigue, most agree that both pathways are likely integrated (Perrey et al., 2010; Roelands and Meeusen, 2010).

Successful muscle activation depends on the propagation of an action potential over the entire sarcolemma and down the transverse-tubules (t-tubules). The signal is transferred from the t-tubules to the sarcoplasmic reticulum (SR) during the excitation-coupling process, resulting in the release of calcium (Ca2+). Elevation of intracellular Ca2+ causes activation of the actin-myosin complex leading to contraction. A decrease in Ca2+ availability from the SR would consequently cause a decline in the binding of actin and myosin to form cross-bridges (muscle contraction; Westerblad et al., 2010), which in turn will cause a gradual decline in force generating capacity. Various intracellular changes can also depress the sensitivity of troponin for Ca2+ and thereby affect performance in the same way as Ca2+ release. Factors that interact with the actin-myosin cross bridging will reduce the power output of each cross-bridge turnover, thus causing fatigue.

The causes of peripheral fatigue are postulated to involve several mechanisms and impairments within the muscle itself, leaving the muscles unable to meet the increased energy demand (Allen et al., 2008; Bishop, 2012). These include: substrate depletion due to metabolic factors (e.g. adenosine triphosphate [ATP], inorganic phosphate [Pi], phosphocreatine [PCr], lactate); ionic factors (e.g. Potassium [K+], Sodium [Na], Ca2+) including disruption of Ca2+ release and uptake within the SR; diminished glycogen availability; acidosis; hypoxia; and micro-trauma within the muscle (Coggan and Coyle, 1991; Allen et al., 2008; Knicker et al., 2011; Bishop, 2012). These mechanisms can subsequently lead to a reduced force/power output by a single muscle cell or motor unit. Where several motor units are impaired, the function of the whole muscle could be adversely affected resulting in diminished exercise performance (Knicker et al., 2011).

Alghannam (2012) illustrated (Figure 2.2) the proposed factors that may contribute to fatigue during a football match. The dashed arrows indicate an unlikely cause of fatigue

and the closed arrows indicate possible causes of fatigue in football. The diagram shows that reduced glycogen stores, dehydration and increased muscle temperature are more probable causes of fatigue towards the end of a game as opposed to a decreased Ca+ availability. As for transient (temporary) fatigue, the diagram illustrates that only increased levels of K+ in the muscle interstitium are a possible cause of fatigue, and lactic acid, muscle power of hydrogen (pH) and ATP-PCr stores being an unlikely cause of transient fatigue.

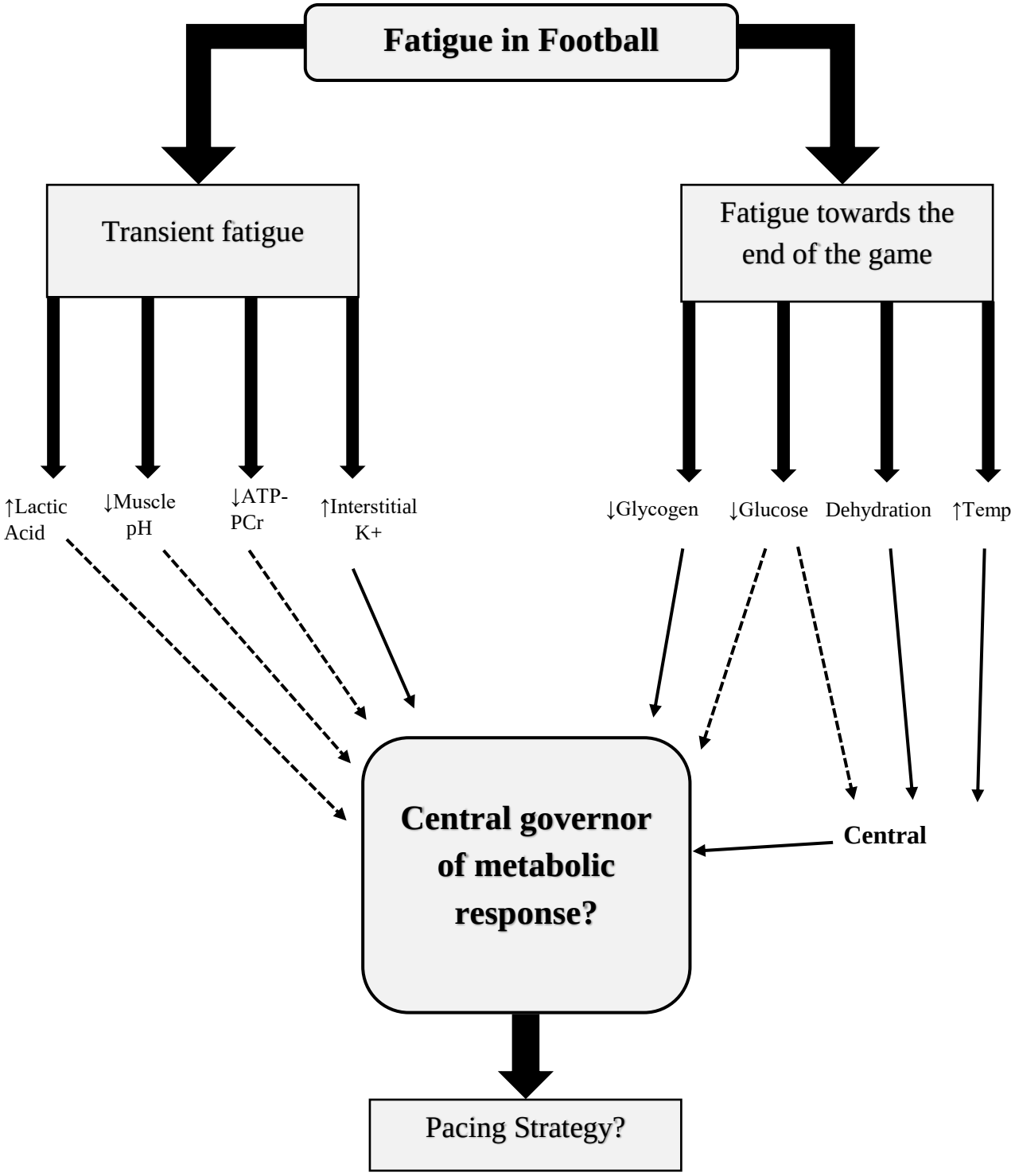


Figure 2.2: Flow diagram illustrating the proposed caused of fatigue occurring transiently and towards the end of a football match. Dashed arrows indicated an unlikely cause of fatigue. Closed arrows indicate possible causes of fatigue in football (Alghannam, 2012).

2.3.2: Transient Fatigue

Temporary fatigue during match-play is known as 'transient fatigue' (Bangsbo et al., 2006; Alghannam, 2012). Transient fatigue is caused by high-intensity anaerobic manoeuvres with short periods of recovery. These repeated high-intensity manoeuvres of accelerating, sprinting, duel play, jumping and tackling form crucial events in football (Di Salvo et al., 2009). Even at the highest standards of competition, players show susceptibility to transient fatigue through a reduction in the total distance and HIR covered after intense periods of activity (Carling et al., 2008; Reilly et al., 2008; Bradley et al., 2010; Bradley et al., 2013). Bradley et al. (2009) demonstrated that high intensity running could be reduced by 50 % to levels below the group mean after the most intense five-minute period during the game, thus indicating the presence of transient fatigue. The dominating metabolic pathway for supplying energy for high intensity, explosive, short bouts of activity is through the anaerobic metabolism (ATP and PCr), depletions of intra-muscular ATP-PCr stores may be a causative factor of transient fatigue (Westerblad et al., 2010; Alghannam, 2012). Following football match-play, individual fibre levels of PCr are almost fully depleted (Krustrup et al., 2006). However, the quick re-synthesis rate of PCr (15-30 s) in the muscle means this is an unlikely cause of temporary fatigue during football. Alternatively, some researchers have suggested that reductions in match running performance could be due to players employing conscious or subconscious pacing strategies to enable them to complete a match without failure of a single physiological system (Drust et al., 2007; Edwards and Noakes, 2009). A drop in HIR running after peak periods could also be influenced by tactical aspects of the game such as changes in ball possession or recovery runs back into position (Bradley and Noakes, 2013; Paul et al., 2015). In research, examining physical performance/fatigue often uses a reductionist approach with limited insight into a player's mental cognition that may interact with processes that limit physical ability. As such, reductions in HIR could easily

be viewed as a player preserving his or her physical readiness for when the game demands increase (Paul et al., 2015).

Intramuscular factors appear to dominate during strenuous activities of high intensity, thus resulting in peripheral fatigue to be a high contributing factor for transient fatigue as it relates to the factors which occur within the muscle that lead to impaired contractile function (Westerblad et al., 2010). However, there is minimal evidence to support that depleted PCr stores cause fatigue during a game (Bangsbo et al., 2006). Other proposed factors linked to transient fatigue are lowered muscle pH levels (muscle acidosis) and increased levels of lactate (Alghannam, 2012).

The production of energy (ATP and pyruvate) for muscle contractions during exercise is a process known as glycogenolysis. In the presence of oxygen (O_2), pyruvate undergoes oxidation in the mitochondria of the muscle fibre to generate more ATP. However, when exercise intensity is high, the breakdown of glycogen via anaerobic glycolysis leads to an intracellular accumulation of inorganic acids, specifically that of lactic acid. Lactic acid dissociates into lactate and hydrogen (H+) and this increase in H+ leads to a reduced pH/muscle acidosis (Westerblad et al., 2002, Westerblad et al., 2010). A reduction in muscle pH is believed to cause a decrease in the binding of Ca2+ to troponin, thereby reducing actin-myosin cross bridges in muscle contraction. The additional side effect of an increase in H+ concentration itself may inhibit actomyosin ATPase directly and lower cross-bridge tension (Metzger and Moss, 1990; Sahlin, 1986). The lactate threshold represents the point at which lactic acid concentration in the blood will begin to increase faster than the rate at which it can be removed. During football matches, average blood lactate concentrations have been reported between 2 and 12 mmol/L, with levels peaking towards the end of each match half. Furthermore, lactate concentrations during the

second half were lower in comparison to the first, suggesting a potential decrease in time spent above lactate threshold as players begin to tire (Bangsbo et al., 1991; Krustrup et al., 2006). Krustrup et al. (2006) noted a weak correlation between reduced sprint performance and lactate accumulation, although this was found to be non-significant. However, the absolute increase in muscle lactate was to moderate values (15.9 to 16.9 mmol/kg/d.w). Nevertheless, muscle pH measured during a football match was only transiently reduced (muscle lactate < 6.8 mmol) and no association with performance decrement was observed (Krustrup et al., 2006).

Until recently, muscle pH was considered an important cause of impaired contractile function in muscle fatigue, yet recent studies have shown that muscle acidosis may have little direct effect on contraction in mammalian muscle at physiological temperatures (Allen et al., 2008; Westerblad et al., 2010). The current evidence in favour of muscle acidosis causing reduced force production comes from studies on skinned muscle fibres and often performed on animals (Wiseman et al., 1996; Westerblad et al., 1997). Therefore, this puts into question the relevance of these findings to fatigue in humans (Westerblad et al., 2002). A review by Westerblad et al. (2002) concluded that muscle acidosis might have an indirect effect on muscle fatigue, such that the activation of certain nerve afferents in a muscle (group III-IV) may lead to the sensation of discomfort experienced during fatigue. The review also suggested a coincidental rather than causal relationship between declining pH and reduced muscle function. That is, with high intensity activity the energy demand exceeds that of aerobic metabolism and so anaerobic pathways are used to generate ATP that in turn increases the production of lactic acid. It is more likely that other consequences of anaerobic metabolism are the cause for impaired contractile functioning over that of muscle acidosis.

Mohr et al. (2005) concluded, in his review of the literature, that temporary fatigue is not directly linked to high muscle lactate, high muscle acidosis or low PCr. The primary suggested factor for the cause of transient fatigue has been linked to an accumulation of K+ in the muscle interstitium, which if high enough can depolarise the muscle membrane potential (Mohr et al., 2005). After exhaustive exercise, blood K+ concentrations have been seen to rise to 11 mmol/L (Mohr et al., 2004; Nielsen et al., 2004), which may be high enough to cause electrical disturbances and reduce muscle recruitment and as a result force development (Cairns and Dukhunty, 1995). The proposed mechanisms for this accumulation of K+ is said to occur through the opening of ATP-sensitive K+ channel (kATP) channels located in the sarcolemma, which tend to open when intramuscular pH declines (Davies 1990; Davies et al., 1991; Bangsbo et al., 1996; Mohr et al., 2005). A simultaneous link in increased interstitial K+ and a decline in muscle pH has been observed in exhaustive exercise (Bangsbo et al., 1996; Nordsborg et al., 2003). These findings therefore support an indirect link between muscle acidosis and transient fatigue. Nevertheless, research is limited regarding K+ turnover in the muscle during a football game (Mohr et al., 2005; Krustrup et al., 2006) and sufficient information cannot be provided about the accumulation in the muscle interstitium (Krustrup et al., 2006) or the contracting muscle fibres due to the methodological processes undertaken (Bangsbo et al., 2006).

The last proposed contributor to transient fatigue is the hydrolysis of PCr to creatine and Pi from anaerobic metabolism. A rise in Pi has demonstrated a relationship with decreased force production (Allen et al., 2008), a decrease in Ca+ sensitivity and a decrease in SR Ca+ release (Westerblad et al., 2002; Murphy et al., 2004). Most models of cross-bridge action propose that Pi is released in the transition from low-force (weakly attached sites) to high-force (strongly attached sites). Research implies that as Pi

increases during fatigue development, fewer cross-bridges would be in high-force states and force production would decrease (Westerblad et al., 2002). Westerblad et al. (2002) and Allen et al. (2008) discuss the various ways Pi impacts Ca2+ release and handling, however evidence for the above comes mainly from experiments on isolated animal muscle preparations performed at low temperatures, which therefore limits the applicability of such findings to fatigue observed in humans.

A failure anywhere in the sequence of excitation-contraction coupling in muscle could contribute to fatigue (Allen et al., 2008). The intramuscular metabolic changes as discussed above can adversely affect the activation of skeletal muscle. Such changes may proceed to decrease myofibrillar Ca2+ sensitivity, reduce SR Ca2+ release and consequently decline the binding of actin and myosin that form cross-bridges for muscle contraction (Westerblad et al., 2010). It therefore seems logical that there may be a close relationship between metabolic changes within the muscle, anaerobic metabolism and transient fatigue. This means some consequences of anaerobic metabolism cause the decline seen in contractile function, but a direct link between these factors is yet to be found in mammalian muscles.

2.3.3: Post-Game Fatigue

The capacity of football players to perform high-intensity efforts declines towards the end of a match regardless of playing ability (Mohr et al., 2003; Bradley et al., 2010; Alghannam, 2012). Research in the English Premier League and from European teams have demonstrated that during the last 15-min of a game the distance covered at high-intensity was 18 to 20 % lower than the first 15-min period of the game (Bradley et al., 2009; Bradley et al., 2010). Bradley et al. (2010) only found the positions of wide

midfielders, central midfielders, fullbacks, attackers, and central defenders applied to this decline in high-intensity effort observed. This deterioration in HIR and repeated sprint bouts has been suggested as an outcome of fatigue. Interestingly, substitutes who came on in the second half performed more sprints and high intensity runs (63 and 25 %, respectively) than players who played the entire game. However, the exact reasons for this end of game fatigue is uncertain. Research has focused on decreased glycogen stores, dehydration and increased muscle temperature. Other contributing factors such as tactics, style of play, ball possession and technical qualities cannot be ruled-out as an attribution to a reduction in physical performance (Gregson et al., 2010).

Immediately post-match in elite female soccer, decrements of up to 62 % have been found in the Yo-Yo Intermittent endurance level 2 (Yo-Yo IE2) test in comparison to testing in a rested state (Krustrup et al., 2010). There were also reductions in the average 30 m sprint time, shown to be 4 % slower post-match. Interestingly, vertical jump performance (height) was unaffected immediately post-match. The extreme drop in Yo-Yo IE2 emphasises the reduced recovery capacity during sustained intense exercise bouts with short recovery periods similar to that experienced during competitive match-play. Such decrements in physical performance help to explain the type of fatigue present immediately post-match that may be attributable to lowered muscle glycogen levels. Vertical jump performance may have been unaffected immediately post-match due to the athlete's capacity to change their movement mechanics to sustain peak outputs in one explosive movement (Andersson et al., 2008). Nevertheless, decrements in jump performance may have been more evident in the following 24-48-h post-match where low-frequency fatigue related to the process of repair and regeneration becomes predominant in muscle tissue (Jones, 1996; Keeton and Binder-Maleod, 2006).

Glycogen is the main source of energy for muscle contractions (Hermansen et al., 1967). Glycogen serves as a storage form of glucose and is abundant in skeletal muscles and the liver (Ørtenblad et al., 2013). A depletion in glycogen stores is said to result in a reduction of ATP regeneration, which in turn leads to impaired contractile function due to the muscle being unable to maintain the processes involved in excitation-contraction coupling (Ørtenblad et al., 2013). Impaired contractile functioning causes a reduced force output, a sign of muscular fatigue (Reilly et al., 2008; Bishop, 2012). Earlier studies argued that muscle glycogen would not be depleted enough to affect the maintenance of glycolytic rate when almost depleted by 50 % (Jacobs et al., 1982; Bangsbo et al., 1992). Nevertheless, when investigating single muscles fibres, it was noted that individual muscle fibres, particularly fast twitch, were depleted or partially depleted of glycogen. Such depletion in individual fast twitch muscle fibres does not allow maximum glycolytic rate to be maintained and maximum muscle contraction can therefore not be performed. Consequently, reduced high intensity and sprint efforts may be observed in the last 15-min of a game (Bangsbo et al., 1992). Studies investigating muscle glycogen concentration after a football match demonstrate significant decreases (Krustrup et al., 2006; Krustrup et al., 2011). Furthermore, Krustrup et al. (2011) examined the time course of muscle glycogen content over 72-h in seven professional football players from the Danish first and second divisions. Post-match biopsies obtained from the *vastus lateralis* muscle revealed glycogen content to be only 43 % of the pre-game concentration. At 24-h, glycogen levels had partially recovered but remained 27 % lower than pre-match values. The time course of complete glycogen replenishment for football players following a match appears to be between 24-h and 72-h (Krustrup et al., 2006; Bangsbo et al., 2006). This time course of replenishment links to the residual signs of physical fatigue which are evident up to 72-h post-match (Mohr et al., 2003; Andersson et al., 2008; Ispirlidis et al., 2008; Fatouros et al., 2010). To avoid future decrements in

performance, the timing and the type of carbohydrate (CHO) ingestion post-exercise should be considered (Zehnder et al., 2004).

Studies by Duhamel et al. (2006a, and 2006b) and Ørtenblad et al. (2011) have linked the role of glycogen availability to SR Ca2+ handling and fatigue towards the end of a game. Duhamel et al. (2006b) manipulated muscle glycogen concentrations with four days of low and high CHO diets to observe the direct effects on muscle glycogen stores and SR Ca+ release. His findings showed a low CHO diet lead to reduced muscle glycogen stores at the initiation of exercise and earlier deterioration in SR Ca2+ release in comparison to the high CHO diet. Carbohydrate supplementation has also been shown to lessen exercise-induced changes in blood-based measures (such as free fatty acids [FFA], branch-chain amino acids and ammonia) which have previously been associated to central fatigue (Davis and Bailey, 1997; Newsholme and Blomstrand, 2006). These findings demonstrate the importance of understanding muscle fatigue to help with the correct nutritional diet to be implemented to optimise post-exercise recovery and evade early onset of fatigue during exercise.

Ørtenblad et al. (2011) found similar findings in a cross-country skiing exercise when altering CHO intake 4-h post-exercise. Those skiers with an enriched CHO-diet showed increased muscle glycogen content and a normalised SR Ca2+ release rate. These findings suggest that the SR vesicle Ca2+ release rate is associated with a specific pool of glycogen in the intermyofibrillar compartment (Ørtenblad et al., 2011). Together these studies provide evidence in favour of a direct role between decreased glycogen within the muscles and a reduction in SR Ca2+ release during fatigue. Although studies demonstrate the failure of SR Ca2+ release during fatigue, there is much debate as to whether this occurs through changes in the degree of voltage sensor activation, through

depletion of Ca2+ inside the SR or through the influence of changing myoplasmic metabolites (Allen et al., 2008). If Ca2+ release is reduced by the SR, this in turn would reduce the myoplasmic free Ca2+. If Ca2+ is not present within the myoplasm to bind to troponin C, tropomyosin will be blocking the myosin binding sites on actin and cross-bridges will be unable to form. The inability to form cross-bridges will eventually result in reduced force development (Allen et al., 2008). It is therefore plausible that the decrement in football performance observed towards the end of a game is, at least in part, attributable to exercise-induced reductions in important energy substrates (such as blood glucose, liver and muscle glycogen) for both muscle and central nervous function.

As concentrations of glycogen diminish, FFA are seen to increase during a football-match, with increased values observed at half-time and post-match (Krustrup et al., 2006). This increase may represent the elevated uptake and oxidation of FFA to the contracting muscle consequently maintaining blood glucose. Evidence of such metabolism further suggests the reduction in glycogen content of muscle tissue.

Other proposed contributors to fatigue towards the end of a game are dehydration and an increase in muscle temperature, better known as hyperthermia (Mohr et al., 2010). It has been shown that players can lose more than 2 to 3 litres (L) of fluid during a game in a normal thermal environment and up to 4 to 5 L of fluid in a hot humid environment (Bangsbo, 1994; Reilly, 1997; Shirreffs et al., 2006; Alghannam, 2012). Decrements in a sport-specific fitness test post-match demonstrated 13 to 15 % decrease in distance covered where fluid ingestion had been prevented in participants (Edwards et al., 2007). Conversely, other studies have shown no adverse effects on anaerobic performance or technical ability in participants with moderate fluid loss (Hoffman et al., 1995; Cheuvront et al., 2006).

Exercise fatigue or a decline in performance may occur more rapidly at higher temperatures (Allen et al., 2008). If the body temperature fluctuates outside of a narrow, optimal range, there are small physiological changes within the body to allow for homeostasis. Physiological temperature regulation is known as thermoregulation and includes heat flow via the blood from the core to the skin, sweating (for evaporative heat loss), and metabolic heat production (e.g. shivering; Kenefick et al., 2007). In higher ambient conditions, players' overall work-rate and high intensity activity is adversely impacted. Mohr and Krustrup (2013) demonstrated that heat stress in a player could impair the repeated ability to perform crucial anaerobic components of the game. Moreover, a fluid loss of 1 to 2 % of body mass could result in a rise in core temperature (Alghannam, 2012). As such, dehydration and hyperthermia may come hand in hand when contributing to the intricate phenomenon of fatigue in football. It is likely that these two factors may be major components in the cause of central fatigue (Gonzalez-Alonso et al., 1999; Nybo and Nielsen, 2001). It has been stated that heat exhaustion in humans seems to occur at a core temperature of 40°C. Maughan et al. (2010) found a number of players to exceed a core temperature of 40°C, which may instigate central fatigue and cause a decline in muscle performance due to inadequate recruitment of motor neurons (Nybo and Secher, 2004). Nevertheless, Mohr et al. (2004) found no differences in core temperature between the end of each half and during a game, and core temperature did not exceed 39°C. These findings would suggest that a rise in core temperature during a game cannot be consistently established as a cause of central fatigue.

In summary, exercise fatigue is recognised as a complex, multifactorial, task-dependent factor whose aetiology has been much debated among experts in the field (Enoka and Duchateau, 2008). Research into exercise fatigue is abundant, yet the exact cause of fatigue remains to be established. The process of muscle contractions involves several

steps beginning with an electrical impulse sent from the motor cortex in the brain and ending with the formation of cross-bridges from the mechanical binding of actin and myosin. Almost every stage of this process can be disrupted, thus any prolonged physical activity performed at enough muscle intensity will produce fatigue and impair the capacity of the neuromuscular system to generate the required force (Gibson and Edwards, 1985). The causes of fatigue in football remains uncertain with numerous contributing factors to both transient and end of game fatigue. Obtaining knowledge of all probable aspects to fatigue is imperative, because if fatigue levels are poorly managed there may be detrimental outcomes for players, such as decrements in performance and increased injury risk (Twist and Highton, 2013). Therefore, practitioners in elite sport aim to reduce and evade the effects of fatigue in order to achieve the highest possible levels of performance on the pitch.

2.4: Mechanical Muscle Damage and Soreness

Intense actions seen in elite football such as accelerations, decelerations, sprinting, striking, tackling and jumping can result in non-metabolic fatigue from disruption to internal structures mediated by these high force actions. Tissue damage from such repetitive actions can impair muscle function (Hubal et al., 2007; Hedayatpour et al., 2009) and consequently affect the performance of athletes. Muscle damage and muscle soreness can be caused by strenuous or unaccustomed exercises, and particularly eccentric muscle contractions, that may be present for up to 72-h (Ispirlidis et al., 2008; Fatouros et al., 2010). An eccentric muscle contraction is whereby the muscle lengthens as it develops tension (Appell et al., 1992). In relation to football, this may be when the player performs cutting movements, decelerations and changes of direction. The possible mechanisms of muscle damage from eccentric/unaccustomed exercises are well

documented (Byrne et al., 2004), but the exact reasons as to why it causes muscle soreness remains to be elucidated (Pyne, 1994; Nosaka et al., 2002; Butterfield et al., 2006).

Eccentric force production is essential when decelerating as the hip, knee and ankle extensors all work eccentrically to increase the body's braking forces. Failure of these muscles to produce the required force to adequately decelerate the body may lead to physical performance being compromised during stopping or changing direction, resulting in an increased risk of injury (Smith et al., 2009). Structurally, eccentric exercises potentially lead to focal myofibril disruption and loss of subcellular Z disc integrity and have been shown to typify the structural damage to skeletal muscle and may be accompanied by a leak of intracellular proteins into the bloodstream (Proske and Morgan, 2001; Magal et al., 2010; Raastad et al., 2010). This loss of structural muscle integrity is said to cause secondary damage associated with subsequent inflammatory response and infiltration of neutrophils that further, in isolation, compromise the mechanically damaged area (Butterfield et al., 2006). The characteristics of such muscle damage involve a temporary reduction in muscle function, an increase in perceptual muscle soreness and evidence of swelling (Howatson and van Someren, 2008). Mechanical muscle damage derived from actions with a high eccentric component may therefore contribute to a reduction in physical performance observed during and after a football match.

At the end of a football match, elevated levels of intracellular proteins such as CK have been reported in elite and sub-elite players (Ispirlidis et al., 2008; Fatouros et al., 2010; Thorpe and Sunderland, 2012; Silva et al., 2013). These elevated levels of intracellular proteins have been used as indirect markers of muscle damage to quantify the extent of

muscle damage imposed by matches and training (Mougios, 2007; Coelho et al., 2014; Hody et al., 2013). The time-course of potential muscle damage markers immediately and in the hours and days following a competitive football match will be discussed later in this review.

2.5: Training Models Applied to Football

To control the level of fatigue imposed on a player, an understanding on the dose-response relationship is required, such as the positive (improved cardiovascular fitness) and negative (increased fatigue) consequences of the daily individual training sessions (Kellman et al., 2018). The Supercompensation Model (and/or General Adaption Syndrome) is the most comprehensible representation of the training adaptation response. It is a concept that underpins almost all sports coaches' and sports scientists' planning and management of training programs for elite athletes. Individual players respond differently to the physiological stress of exercise, with Hans Seyle proposing the General Adaptation Syndrome (GAS) in 1956. The GAS theory describes how each individual responds to a stressor. Initially there is an 'Alarm stage', which involves the negative response of stress. In relation to training, this may take the form of delayed onset of muscle soreness (DOMS), fatigue or stiffness. Next is the resistance or 'Supercompensation' stage where positive adaptations occur. During this stage the body will reorganise and adapt its capacities so that the next exposure to the same stimulus will have a reduced stress response. Supercompensation can only be achieved after a period in which the accumulated fatigue from training can be reduced. The length of time required for regeneration or recovery is primarily dependent upon the magnitude of the initial stressor and subsequent displacement in homeostasis. Lastly is the 'Exhaustion stage', occurring only when the magnitude of a stressor is too large or the accumulation

of stress is too great resulting in maladaptation to occur (Selye, 1950; Kentta and Hassmen, 1998; Chiu and Barnes, 2003; Gamble, 2006; Bishop et al., 2008; Turner, 2011). Inadequate recovery time will lead to an accumulation of stress over periods of days or weeks, which is why accurate monitoring of an individual's response to the load becomes paramount. If an individual's threshold of accumulated fatigue is reached, maladaptation from training can occur, resulting in performance decrements and a state of overtraining.

The Fitness Fatigue Model (FFM) proposed by Bannister in 1975 is a development of the GAS theory, comprising a better representation of stimulus and response. It argues that different types of training stressors generate different levels of physiological response. Each type of training results in simultaneous fitness and fatigue responses, which can positively or negatively affect performance (Chiu and Barnes, 2003). When the fitness response outweighs the fatigue response, it gives rise to an improvement in performance. It is suggested that the after-effects of fitness and fatigue are exercise specific. For example, they may be neuromuscular or metabolic and if one of these systems has been fatigued, the other may still be able to perform exercises to satisfaction. In this aspect, this rationalises how training would incorporate both resistance and aerobic workouts (Turner, 2011).

In relation to football, the GAS theory and Fitness Fatigue Model both suggest that players can adapt themselves to changes in training type and intensity. They also demonstrate the importance of monitoring training and fatigue responses to avoid overtraining and to help increase the preparedness of players. However, the magnitude of adaptability to a stressor is said to depend largely upon genetic factors (Selye, 1950). Genes control every aspect of an individual, including the foundations of an athlete such

as muscle, cartilage and bone formation, muscle energy production, metabolism, and blood and tissue oxygenation (Kambouris et al., 2012). If genetic factors have a role in determining sports performance, it questions why some clubs operate a group approach in the management of a football team. Specific pathways for post-exercise recovery could be utilised by sub-grouping players depending on their individual response to exercise. Continual monitoring of individual responses to load will help avoid an athlete's critical point or threshold where their reserve capacities cannot cope with the accumulated fatigue (Kentta and Hassmen, 1998). To evade the 'exhaustion phase' where overtraining, performance decrements, and injury may occur, an optimal training program needs to assess the individual athlete's current tolerance to stress or fatigue from a training and a competitive performance perspective (Urhausen et al., 1995).

2.6: Fatigue Management

In elite sport, practitioners are taking an ever-increasingly scientific approach in the monitoring of fatigue through training load (Djaoui et al., 2017; Carling et al., 2018). Fatigue can be managed through planned alterations of loading and unloading in a structured cyclic manner (Gamble, 2006; Turner, 2011; Mara et al., 2015). This concept, known as periodisation, offers a framework for planned systematic variation of training parameters to ensure the level of performance is kept consistently as high as possible and fatigue levels are managed to elicit adaptation (Gamble, 2006). This cyclic structure works alongside the GAS theory and FFM in order to safely overload players and provide taper periods to allow for positive adaptations so that an athlete's fitness outweighs their fatigue response.

Competition frequency places a heavy influence on the in-season micro-cycle in football, which typically lasts three to seven days in duration (Kelly et al., 2020). The micro-cycle will encompass fluctuations in training volume and intensity to allow players to perform at a high level on match day (Bangsbo et al., 2006). In general, there are observable decreases in training volume as match day approaches to facilitate decline of fatigue (Clemente et al., 2014, Kelly et al., 2020). With an amplification in game and training demands there comes an increased need to monitor the associated fatigue responses, since maximising the adaptive response to training is also reliant on avoiding the negative consequences of excessive fatigue. While designing structured training programmes appears to be the first step in performance management, the second most important step towards maximising an adaptive response is monitoring the impact of training and games on players. Therefore, if needed, adjustments could be made to the training regimen and overtraining could be avoided, and the occurrence of illness and injury could be reduced (Drew and Finch, 2016).

The management of player load from training has been proposed as a central contributor to reducing subsequent injury risk (Gabbett and Domrow, 2007; Brooks et al., 2008; Rogalski et al., 2012). The relationship between training load and injury rates has previously been reported (Gabbett, 2004; Gabbett and Jenkins, 2011; Gabbett and Ullah, 2012), and suggests that the harder individual trains, the higher the likelihood of injury and illness in elite athletes. Gabbett and Jenkins (2011) demonstrated that a higher training workload (rating of perceived exertion x session duration) was related to overall incidence of injury ($r = 0.82$, $P < 0.01$), for both contact and non-contact injuries ($r = 0.80$ to 0.82, $P < 0.01$) in 79 professional Rugby League players. In a more extensive study of 220 sub-elite rugby players the incidence of injury was significantly higher in the 2001 pre-season period (156.7 per 1000 training h, 95 % CI 136.3 to 177.1) where

training duration and intensity was higher in comparison to the subsequent 2002 and 2003 pre-seasons (78.4 to 94.4 per 1000 training h, 95 % CI 64.2 to 112.0). However, in contrast, too little workload has been shown to elicit a detraining response and increases the inherent risk of injury (Gabbett and Domrow, 2005). Therefore, research indicates a delicate balance between the minimum workload required to elicit an improvement in performance and the maximum workload tolerable before increasing injury risk (particularly of soft tissue nature).

The acute:chronic workload ratio has become of popular use for observing spikes in workload over time. The acute:chronic workload ratio refers to the workload performed in one week (acute workload) relative to the four-week chronic workload (four-week average acute workload: Banister et al., 1975; Banister and Calvert, 1980). Hulin et al. (2016) observed acute:chronic workload ratios over the course of two seasons in elite Rugby League players. An innovative finding exposed that a high chronic workload combined with a moderate, and moderate-high workload ratio had a smaller risk of injury than a low chronic workload combined with different acute:chronic workload ratios. These findings demonstrate that a steady increase in acute workload will subsequently increase the chronic workload, while a high chronic workload can protect against injury when acute workload is similar to the chronic workload.

Training and match demands can be quantified objectively by player tracking systems (global positioning devices, multiple-camera computerised tracking system) and heart-rate monitors. Di Salvo et al. (2006) validated the use of Prozone® ® Sports Ltd., video-motion analysis tool, reporting that the system provided an accurate estimation of different movement velocities performed by athletes on the pitch. The generation of physical match performance data using Prozone® is based upon the development of a

continuous movement trajectory for each individual player during match-play (Di Salvo et al., 2006). Previous studies have evaluated the validity and reliability of Prozone® and found the system to be reliable for measuring match activity in football (mean CV of 16.2 ± 6.4 %, 95 % CI = 15.6 to 16.7: Di Salvo et al., 2006; Di Salvo et al., 2009; Gregson et al., 2010).

Global Positioning Systems (GPS) allow users to accurately determine positions on the surface of the earth from a network of satellites orbiting around the planet. Over the past ten-years, there has been a rapid uptake of GPS technology within football to track speed of movement and distance covered during training sessions and match-play (Aughey, 2011; Bastida et al., 2018). Rampinini et al. (2015) compared two high-frequency GPS units (5-Hz and 10-Hz), with a radar system (Stalker ATS, Radar Sales, Minneapolis, MN, US) considered together with the laser systems the gold standard for the measurement of instantaneous speed. Only the 10-Hz GPS unit displayed a sufficient level of accuracy for quantifying distance covered at higher speeds. To determine the validity and reliability of measurements, the classification of GPS into different frequencies is necessary and provides information as to which is the most appropriate GPS to apply under different sport data acquisition conditions.

Through these systems, pracritioners in elite sport are able to continuously collect objective data to allow ample information on player and team physical performance data by providing parameters such as distance covered, accelerations, decelerations and velocities. The use of this data can allow training targets to be set on a daily/weekly basis (micro-cycle). This micro-cycle would encompass specific days of high, and low loading prior to an upcoming fixture with the aim of peaking player performance ready for match-day. In addition to objective measures of training demands, physiological and physical

performance testing can be conducted to observe the recovery-fatigue status of players from training and competition. These objective measures can be used collectively to observe for over overtraining, thus providing a holistic approach to athletic monitoring and fatigue management.

2.7: Metabolic Load and Fatigue

An area often overlooked when monitoring external training load from GPS technology is the frequency of activities related to accelerations and decelerations (Akenhead et al., 2016). High intensity energy bouts such as changes in direction, accelerations and decelerations are frequently performed in team sports and elicit high metabolic and neuromuscular demands (Varley and Aughey, 2013; Hodgson et al., 2014; Akenhead et al., 2016). Within football match-play over 18 % of the distance covered is attained while accelerating or decelerating (Akenhead et al., 2013). Such actions impose great physiological demands on athletes over that constant linear running (Varley and Aughey, 2013). During football match-play, high intensity acceleration and deceleration capacity is reduced and compromised as fatigue increases. These reductions have shown to be sensitive in the observation of fatigue, more so than that of HIR and SD (Akenhead et al., 2013). This increased sensitivity is facilitated by the number of maximal accelerations performed in a game being eight-fold greater than that of sprints (65 ± 21 vs. 8 ± 5: Varley and Aughey, 2013).

Akenhead et al. (2013) examined low, moderate and high acceleration and deceleration during competitive football games. The finding of most relevance, to fatigue, was that high intensity acceleration and deceleration performance following the peak five-minute value was on average 10.4 and 11.4 % lower than the mean values. This observable

decline may be attributable to transient and peripheral fatigue mechanisms. In support of these findings, Arruda et al. (2015) observed GPS parameters during a congested match schedule and demonstrated a decrease in the frequency of accelerations per minute across the competition, with a notably greater number observed during the first match. Further, Dalen et al. (2019) found a greater decline in the acceleration profile of elite football players to that of high-speed running, with 34 % less accelerations from the first to the last five-minute period of a game. The work by Dalen et al. (2019) demonstrated that accelerations may be more sensitive, across all playing positions, as a measure of physical performance decline. Therefore, these results support that the observation of frequency of accelerations/decelerations may be useful to monitor external load during matches and training as it may link to higher levels of fatigue. If an individual player shows a high frequency of accelerations per minute, or a greater reduction towards the end of a match, their subsequent training and recovery could be adapted to compensate for this.

Across the literature, associations have been made between time–motion parameters and the magnitude of skeletal muscle damage reported in Australian Rules football players (McLellan et al., 2011; Young et al., 2012). Young et al. (2012) looked at GPS variables and post-game CK levels. The authors reported that accelerations, decelerations and high intensity running are all large contributors to high CK levels and indicators of muscle damage. Similarly, research by de Hoyo et al. (2016) in elite under eighteen-year-old football players demonstrated a greater volume of high acceleration and deceleration tasks were correlated with CK levels. De Hoyo et al. (2016) further reported a change in CMJ average eccentric force production at 30-min post-match (−3.99 ± 6.32 %), which was associated with the number of decelerations during the match. Decelerations in particular, require intense eccentric contractions of various muscle groups (Young et al.,

2012), and are known to induce muscle damage, as indicated by increased plasma CK (Nosaka and Newton, 2002).

Collectively, the aforementioned research supports the quantification and observation of HIR, accelerations and decelerations in football to provide practitioners with additional information regarding external training load, training/match intensity and subsequent associations with skeletal muscle damage.

2.8: Indirect Markers of Fatigue

Practitioners in elite sports are taking an increasingly scientific approach to monitoring the recovery-fatigue status of athletes. Copious amounts of data are being collected from player testing and this, now, common practice has become an important aspect of sport in recent years (Djaoui et al., 2017; Carling et al., 2018). The rationale for this data collection is to provide an advantageous insight into a player's level of fatigue prior to the next fixture, so if required additional recovery modalities could be provided to reduce the effects of fatigue and enable the player to achieve the highest possible levels of performance.

The majority of field test recommendations in team sports, for standardised performance measurements, are physically demanding and induce additional fatigue (Bangsbo and Lindquist, 1992; Nedelec et al., 2012). Such field tests are not suitable in elite sport, especially during the competitive season, where players have limited recovery time between training and games. Consequently, surrogate physical performance tests and biomarkers are being used to make informed decisions on the recovery-fatigue status of players. Such tests/biomarkers include CK levels, cortisol, testosterone, hydration levels, jump tests and isokinetic dynamometry. Nonetheless, each of the above-mentioned are

subject to individual variability. Factors such as gender, genetic predisposition, mode of muscular actions, the exercise duration/intensity, and distribution of fibre types in skeletal muscles may influence individual scores (Tesch et al., 1985; Heled et al., 2007; Yamin et al., 2007). Therefore, the correct understanding and interpretation of results is fundamental to detect meaningful changes in the recovery-fatigue status of players, and to avoid injury, performance decrements and overtraining (Twist and Highton, 2013; Kellman et al., 2018).

The collection and analysis of biomarkers presents many challenges: (a) a single biomarker is not definitive for diagnosing broad physiological function such as "fatigue" in sport; (b) the sensitivity of single biomarkers to detect overtraining or injury risk is limited; (c) reference ranges for athletes and specific subgroups of athletes (e.g. elite senior football players) are not well defined; (d) there is high inter-individual variance in absolute values and relative changes in biomarkers; and (e) the nature of biomarker testing and analysis is highly contextualised (Lee et al., 2017). Practitioners should take careful consideration of the above mentioned when trying to detect small meaningful changes in an athlete's physical/physiological response to exercise stress.

The development and implementation of insightful field tests/biomarkers that monitor an individual's recovery from intense training or competition may support important decisions on player involvement in both preparation periods and games and is therefore of interest to both practitioners and coaches. The biomarkers and physical performance tests discussed below are those commonly collected in elite sport (de Hoyo et al., 2016; Ascensão et al., 2008; Ispirlidis et al., 2008; Magalhães et al., 2010, Balloch, 2018; Cormack et al., 2008b; Rowel et al., 2018).

2.8.1: Creatine Kinase

CK is a muscle-specific protein in the blood; it is an indirect marker of muscle damage. In sport CK has been used to quantify the extent of muscle damage imposed by matches and training (Mougios, 2007; Hody et al., 2013; Coelho et al., 2014). Creatine kinase is an intra-muscular enzyme required for the phosphorylation of adenosine diphosphate (ADP) to free creatine; due to its large molecular weight (~80,000 g/mol) it is unable to pass directly into the circulation (Magal et al., 2010). After performing strenuous, unaccustomed exercise or eccentric actions there is a mechanical disruption to the cellular structure of muscle (Newham et al., 1986; Proske and Morgan, 2001). Structurally, eccentric exercises may lead to weakened sarcomeres, resulting in membrane disruption and a leak of intracellular proteins, such as CK, into the bloodstream (Proske and Morgan, 2001; Magal et al., 2010). Therefore, an increased level of the plasma CK activity is generally accepted as a good indicator of exercise-induced muscle damage (Fielding et al., 2000; Hartmann and Mester, 2000; Hody et al., 2013).

In team sports such as football, eccentric actions occur intermittently and at a high level of intensity (Coelho et al., 2014). Tissue damage from such repetitive actions can impair muscle function (Hubal et al., 2007; Hedayatpour et al., 2009) and consequently affect the performance of athletes. The observation of CK levels in sport can subsequently be used to detect players with a higher risk of suffering from severe breakdown of muscle tissue (Hody et al., 2013). Furthermore, collecting CK levels may be advantageous for monitoring training load and the over-training status of athletes (Brancaccio et al., 2007; Lazarim et al., 2009). However, the post-exercise plasma CK activity shows a large variation across individuals, which needs to be accounted for (Chen, 2006; Mahmutyazicioglu et al., 2018). The different individual responses in CK has been

seemingly linked to several factors such as genetic background (Clarkson et al., 2005; Yamin et al., 2007), physiological and morphological makeup (Lee and Clarkson, 2003; Heled et al., 2007).

Despite the common observation of CK as a marker of muscle damage, there have been questions regarding its validity, especially when trying to establish the magnitude and temporal sequence of damage (Friden and Lieber, 2001). Fielding et al. (1993) reported there was a lack of association between ultra-structural damage determined through z-band disruption and plasma CK concentrations. Cobley et al. (2011) documented that athletes with the highest CK values did not necessarily indicate the highest levels of muscle damage. Further, it is plausible that the efflux of intracellular proteins, such as CK, results from both structural damage and transient changes in membrane permeability (Brancaccio et al., 2007). These findings indicate that CK should not be used to quantify the magnitude of muscle damage and/or be used to compare damage between individuals. Such research also questions how much cellular muscle damage needs to occur before there is a marked increase in CK concentration.

In athletes, it has been found that CK concentrations are higher at rest compared with untrained subjects and concentrations have been shown to stay elevated for several days after a prolonged or intense training session (Mougios, 2007). Table 2.1 shows the CK reference intervals in professional football players as stated in the current research. Many professional athletes train daily which will also account for a higher average CK levels than in untrained individuals (Branccaccio et al., 2007). Studies have reported CK levels to peak 12-h to 48-h post-match and remain elevated for up to 72-h (Coelho et al., 2011). These elevated levels have corresponded with decrements in physical performance testing such as CMJ scores and sprint ability (Ispirlidis et al., 2008; Magalhaes et al.,

2010; Coelho et al., 2011; Hunkin et al., 2014). The testing of CK concentrations following a football match to assess for signs of muscular fatigue/stress has previously been investigated (Table 2.2).

Table 2.1. Mean CK reference values for football players.

Author	Mean CK value (U/L)
Ispirlidis et al. (2008)	**520**
Scott et al. (2016)	**520**
De Hoyo et al. (2016)	**358**
Burgess et al. (2016)	**950**
Hecksteden and Meyer (2020)	**343**

Table 2.2. Summary of studies undertaken on recovery time-course of plasma CK (u/l) in football.

					Mean ± SD		Time point post-activity			
Author	**Participants**	**Playing level**	**Football activity**	**CK testing procedure**	**Pre-activity**	**0 h**	**Day 1**	**Day 2**	**Day 3**	**Day 4**
De Hoyo et al. (2016)	15 young elite	Spanish first league senior team (U19)	Match	Fingertip capillary	290.24 ± 145.81	+28.6 %	+43.4 %	+26.0 %		
Coelho et al. (2011)	17 professionals	Brazilian first division national championship	Match	Fingertip capillary	192.1 ± 23.0		785.8 ± 95.5 *	388.2 ± 37.8*	299.1 ± 30.5*	317.0 ± 37.7*
Scott et al. (2016)	15 elite	English Premier league	Match (season)	Fingertip capillary				520 ± 224		
Nedelec et al. (2014)	10 professionals		Match	Fingertip capillary	248 ± 105		727 ± 235*	508 ± 210*	470 ± 204*	
Ispirlidis et al. (2008)	24 elite males		Match	Venous blood		+154.3 %*	+400 %*	+710 % (950 u/l)	+637 %	+35.8 %
Russell et al. (2016)	15 Premier league	Premier league (U21)	Match (1-4 games)	Fingertip capillary	334 ± 127		671 (+337 ± 102)	467 (+133 ± 86)		

Blank cells indicate no data reported; Data presented are means or means percentage (%) from baseline; - Decrease | + Increase | * Statistically significant (if stated in research)

Mougios (2007) analysed serum samples from 483 male athletes and 245 female athletes from a variety of sports. The following reference intervals for CK were stated: males 82 to 1083 u/l, and females 47 to 513 u/l. The upper reference limits of football players and swimmers were also stated as 1492 u/l and 523 u/l, respectively. A congested competitive season and the nature of training intensity may contribute to the high reference limits seen in football players. However, a limited amount of research examines the relationship between training intensities and match performance intensities related to CK levels (Scott et al., 2016, Malone et al., 2019; Owens et al., 2019; Hecksteden and Mayer, 2020). Further knowledge regarding the aforementioned and the individual fluctuations post-game would allow for greater indication of individual responses to training/match demands, and a better understanding of potential increased risk of overexertion, and injury.

Coelho et al. (2011) conducted a longitudinal study on post-game CK kinetics in football during the competitive season to observe changes in serum CK at different times post-match. A vast majority of studies focus attention on CK at one time point post-game. This was one of the first studies to objectively analyse CK levels in professional football over four separate time points post-match, ranging from 12 to 110-h. The results demonstrated a peak in CK 12 to 20-h post-match. In addition, they found that maintaining normal training two days post-match might also affect CK removal. They stated that CK can remain elevated up to four days post-match at approximately 300 u/l above baseline measurements. This study identified that high CK levels in the aforementioned range four days post-match can be considered within the norm for football players during the competitive season. Studies such as this support the use of CK for monitoring the training status and recovery of players. However, a limitation to the abovementioned study was the exclusion of game intensity and its impact on CK concentrations. This study also does not state at what time point during the season this data was collected, and there is no mention of high or low loading training weeks. These external factors have previously been shown to influence CK concentrations (Hulkin

et al., 2014, Nowakowska et al., 2019) and exposes areas of research needed to further determine the sensitivity of monitoring fatigue and recovery in football through the observation of CK concentrations.

A congested match schedule and limited player rotations in the team can result in the average pre-match CK value to be 485 % above an individual's baseline (Hunkin et al., 2014). Hunkin et al. (2014) monitored 29 Australian Rules football players at the highest level of competition throughout a season where matches were performed on a weekly basis. A continually high pre-match average CK value was said to be an indication of considerable residual muscle damage throughout the season. In addition to this, CK showed a small negative association with performance ranking scores in inexperienced players - the higher the pre-match CK value the lower the coaches' performance rating for that player in the subsequent match. This study concluded that a sustained elevation in CK likely represents an element of residual muscle damage whereas an elevated CK value in any given week is most likely attributable to inadequate recovery provision from the preceding week.

A study conducted within the Brazilian football championship revealed how the monitoring of CK levels could be used as a practical alternative for early detection of muscle overload. Out of 128 players studied, one player recorded a CK value of 1800 u/l, higher than the upper reference limit of 975 u/l (90^{th} percentile) one day prior to a game and subsequently sustained a muscular overstress injury during that game (Lazarim et al., 2009). Six other players with CK values higher than the reference limit were asked to reduce training for one week, after which they presented lower CK values. The data collected from this study also showed high variability between individuals within the Championship ranging from 200 to 1600 u/l. This supports the large individual responses present in CK values and could be further explained by physical differences between athletes. This research undertaken by Lazarium et al. (2009) further supports the advantageous use of CK for detecting early muscular stress/fatigue,

allowing practitioners to make informed, objective decisions on a players' physiological status and subsequently adapt their training/recovery strategies as necessary.

Overall, an increased level of plasma CK is generally accepted as being a good indicator of muscle damage, but the magnitude of this relationship is controversial (Clarkson et al., 1992; Nosaka and Clarkson, 1996; Fielding et al., 1998; Friden and Lieber, 2001; Cobley et al., 2011). Research in sport suggests collecting CK may be useful for monitoring inadequate recovery and tracking muscle damage across a season. If CK levels remain too high for a particular athlete, training load could be altered to prevent severe muscle breakdown or the development of chronic fatigue. Furthermore, it may query the physical preparedness of certain players pre-match and what potential impact this may have on performance. To observe trends in CK, it is necessary to establish individual baseline measures, given the individual variability in CK, and an individual profile of expected responses over a specific timeframe. Research is limited on the longitudinal trends of this biomarker over a season and its sensitivity to cumulative fatigue in elite football players (Nowakowska et al., 2019). Further investigations into the effectiveness of CK monitoring in the assessment of recovery from muscle damage and its sensitivity to match and training demands would be advantageous. This may provide practical relevance for long-term athlete monitoring by enhancing the implementation of training and recovery modulations to optimise match performance.

2.8.1.1 Individual CK response due to genotype

The characteristic nature of football, as previously discussed, requires players to perform a high number of eccentric actions (Coelho, 2014). Such actions are related to muscle damage and inflammatory responses (Mougios, 2007; Ascensao, 2008; Ispirlidis et al., 2008) and are accountable for the large enzyme efflux observed post-exercise (Newham et al., 1986). To

account for large variability in response to eccentric exercise, some researchers have defined individuals into high, medium and low responders to increases in plasma CK activity (Newham et al., 1986; Clarkson et al., 1992; Chen, 2006). Chen (2006) classified participants' response to eccentric exercises as low (< 500 u/l), medium (500-2,000 u/l), high (2,000-10,000 u/l) and higher (> 10,000 u/l) responders based on their increase in blood CK activity. During this study, participants were required to perform a protocol pre- and post-12-months to evade the repeated bout effect. The difference in CK expression was statistically significant (P = 0.0001) between groups and the results demonstrated remarkably similar individual responses in CK activity between bouts. These findings show consistency in an individual's response to eccentric exercise but research struggles to reason for this individual response rate (Chen, 2006). A clinical definition of a high, low or medium responder is yet to be stated and the underlying reasons for such responses are still unknown (Heled et al., 2007).

Early reports have speculated that type II fibres are more likely to be damaged after a bout of eccentric exercise and therefore may show greater serum CK activity (Thorstensson, 1976; Jansson and Sylven, 1985; Tesch et al., 1985). In contrast, a later study by Magal et al. (2010) looked directly at the relationship between muscle fibre composition and serum CK activity following exercise induced muscle damage (EIMD) in 17 untrained males. In conflict to the earlier studies, the results demonstrated no correlation between the aforementioned. However, the participants were of a homogeneous nature and using a more varied group (power and endurance athletes, sedentary individuals) may have elicited different responses and fibre compositions.

As previously discussed, genetics may be an underpinning factor in an individual's response rate to exercise. Creatine kinase response is more complex than looking solely at fibre type composition and perhaps more attention needs to be given to the morphological makeup of

an individual. In two separate investigations, genetic polymorphism has been linked to CK activity following exercise (Heled et al., 2007; Yamin et al., 2007). Heled et al. (2007) related an exaggerated CK response to a specific CK-MM ncoI gene. Creatine kinase-MM is the muscle specific CK localised at the M-line and the SR of myofibrils. The CK-MM AA genotype was shown to have a six-fold higher increase in CK activity compared to other genotypes (GG and AG). Nonetheless, the underlying mechanism as to why this CK-MM polymorphism may influence CK response to exercise is unknown.

To observe the direct effect of genetic disposition on CK response, Gulbin and Gaffney (2002) investigated 16 pairs of identical twins and their variability in EIMD. Pre-exercise CK concentrations were significantly similar ($r = 0.76$, $P < 0.001$), yet post-exercise the twins did not display the same level of EIMD ($r = 0.15$, $P > 0.05$). This research suggests that the individual variability following high-force eccentric exercise cannot be attributed solely to genetic difference. However, this study tested the elbow flexors and this muscle group is small in comparison to the larger quadriceps or hamstrings muscle group. Testing a larger muscle group would elicit a greater magnitude of response to observe CK activity. Nevertheless, it may be likely that individual response rates to exercise is an interaction between genetic and environmental factors (Chen, 2006). Environmental factors may alter the morphological makeup of twins, leading to different phenotypes and fibre type compositions, which in turn could affect the CK response observed.

The genetic makeup and training history of elite athletes may vary to that of athletes at a lower level or non-athletes and therefore elite athletes may respond differently to training stress and recovery over time (Barnett, 2006). Observing individual player responses to exercise would allow recognition as to which players may need additional support and specific interventions when attempting to accelerate recovery between games. In turn, this would facilitate correct adaptation and supercompensation, whilst avoiding over-training.

With exercise response rates differing between individuals and the high inter-individual variability in CK levels across athletic status, revealing areas of research needed in football to link these two aspects to the recovery-fatigue status of athletes over a competitive season and enhance the physical preparedness prior to a game (Mougios, 2007).

2.8.2: Hydration

The term ‘euhydration’ has been defined as a state of ‘normal’ body water content, a sinusoidal wave that indicates daily water content (Greenleaf, 1992; Oppliger and Bartok, 2002). Research has recommended that athletes should be euhydrated before the commencement of exercise (Coyle, 2004). Exercise stress on the body causes an elevation of metabolic rate that leads to a rise in body temperature if heat loss mechanisms are not invoked. As previously discussed, there are probable links between hypohydration, rises in core temperature, and central fatigue. The imposition of exercise onto daily activity can alter fluid balance homeostasis, performance and health (Cheuvront and Sawka, 2005). Dehydration has been shown to have a negative impact on aerobic and endurance exercise performance through alterations in cardiovascular, thermoregulatory, central nervous system and metabolic functions (Cheuvront et al., 2003). Thus, hydration assessment has become a key component in sport for ensuring full rehydration in athletes frequently performing intense exercise.

2.8.2.1: Fluid Loss after Exercise

Dehydration greater than 2 % body mass should be avoided as this is the threshold after which performance and cognitive function has shown to be reduced (Cheuvront et al., 2003; Coyle, 2004; Lieberman, 2007; Sawka et al., 2007). Edwards et al. (2007) observed differences in post-match performance tests after 11 moderately active male football players

were exposed to three different experimental conditions of 'fluid intake' (FL), 'no fluid' (NFL) and 'mouth rinse' (MR) in a randomised order. The subjects completed a 45-min period of laboratory exercise on cycle ergometers at an intensity equivalent to 90 % of individual ventilator threshold, then participated in a single competitive eight versus eight outdoor football match for 45-min. The finding of most significance was that in both experimental conditions where fluid intake was denied (MR an NFL), the immediate post-match performance during the Yo-Yo intermittent recovery test (YYIRT) was significantly impaired ($P < 0.01$). These two conditions (MR and NFL) also demonstrated post-test losses in body mass (2.1 % and 2.4 %, respectively; $P < 0.01$) indicating that acute, moderate water loss of around 2 % body mass is detrimental to the physiological performance of football players. Although Edwards et al. (2007) showed the importance of fluid intake during football match conditions, it could be argued that if these conditions were applied to professional football players the post-test performance measures may have shown conflicting findings due to the demands of elite football match-play. It could also be debated whether the protocols used in this study were sufficient to elicit transient and end of game fatigue similar to those seen during professional football matches. Further, this study showed a notable increase in core temperature in the NFL condition in comparison to the FL condition during the football match (39.28°C ± 0.35°C vs 38.8°C ± 0.47°C, respectively; $P < 0.05$). It has been suggested that hypohydration is said to be a contributing factor to rises in core body temperature (Gonza´lez-Alonso et al., 1999; Byrne et al., 2006; Alghannam, 2012). However, the core temperatures noted in the study by Edwards et al. (2007) were below the level considered critical to the point of fatigue during prolonged exercise (40 to 40.5°C; Gonza´lez-Alonso et al., 1999; Byrne et al., 2006). Perhaps these small but significant changes in fluid loss and blood plasma concentrations led to a reduction in blood supply to the skin, therefore diminishing the ability to dissipate heat as body water deficit increased (Nadel et al., 1980). These small physiological perturbations within the body could stimulate intrinsic anticipatory mechanisms that invoked performance limitations to avoid

future physical damage (St Clair Gibson and Noakes, 2004). It is likely that hypohydration and rises in core temperature may be a major component in the cause of central fatigue, reiterating the importance of hydration assessment in athletes.

Research by Owens et al. (2013) found inconsistent and limited effects of hypohydration on football passing and shooting skills and high-intensity intermittent running. In contrast with Edwards et al. (2007), they found that performance was not improved in 13 semi-professional, male football players when fluid intake was greater. The most prevalent difference between studies was the levels of dehydration pre and post-testing for the non-fluid conditions. Owen et al. (2013) showed a greater percentage loss in body mass of 2.2 % in comparison to 1.7 % body mass loss in the study performed by Edwards et al. (2007). This variance in body mass loss between the two studies suggests that the decline in high intensity running performance seen in the study by Edwards et al. (2007) cannot be attributable to dehydration alone. However, factors such as the competitive standard of players and the exercise protocol used may have led to these contrasting findings. The protocol used in the study of Edwards et al. (2007), although not directly reflecting a 90-min football match, was more demanding and may have caused a reduction in performance from alternations in the CNS due to higher core temperatures from elevations in metabolic rate. Additional studies have shown that during intermittent exercise, serum sodium, serum osmolality and core temperature are increased during exercise when no fluid is permitted compared to when fluid is ingested (McGregor et al., 1999; Ali et al., 2010). Taking into consideration the aforementioned findings, the cause and effect relationship between fluid loss and rises in core temperature are questionable. It remains unclear whether game-induced dehydration is merely a single characteristic outcome of exercise controlled by a complex metabolic system or a cause of fatigue (Edwards and Noakes, 2009).

2.8.2.2: Hydration Assessment Techniques

There are numerous hydration assessment techniques, and each varies greatly in their applicability due to the methodological limitations. These limitations include factors such as the necessary circumstances for measurement (reliability), sensitivity for detecting small, but meaningful changes in hydration status (accuracy), and the ease and cost of application (simplicity; Oppliger and Bartok, 2002; Sawka et al., 2005). Simple, practical markers of hydration such as urine specific gravity, urine osmolality (Uosm) and colour are said to be reliable assessment techniques to distinguish euhydration from dehydration (Armstrong, 2005), but often correlate poorly with the "gold standard" tests (Popowski et al., 2001). Total body water measurements and plasma osmolality are currently considered the best hydration assessment measures (Armstrong et al., 1994; Cheuvront and Sawka, 2005). These gold standards of hydration assessment are not practical for monitoring day-to-day hydration status of athletes during training or competition due to the analytical expertise and methodological control required (Cheuvront and Sawka, 2005).

Urea is the most abundant protein metabolite in urine, followed by creatinine and uric acid, while electrolytes are the remaining solutes, mainly sodium, chloride, and K+ (Guyton and Hall, 2006). Factors such as increases in muscle mass (Baxmann et al., 2008) or high protein diet (Martin et al., 2006) augment urine protein metabolites concentration, which in turn, could increase urine specific gravity. The measurement of urine specific gravity has a major limitation when testing athletes with a large muscle mass (Hamouti et al., 2010). Hamouti et al. (2010) found a positive correlation between muscle mass and urine protein metabolites ($r = 0.47$; $P = 0.04$) and between urine protein metabolites and urine specific gravity ($r = 0.92$; $P < 0.0001$). Therefore, using urine specific gravity to detect hypohydration may be reduced

in athletes with large muscle mass and therefore other methods of assessing hydration status should be used in elite sport to detect meaningful changes.

In professional football, where time constraints are commonplace, practitioners require testing that is simple and practical, yet accurate enough to detect small but meaningful changes in hydration status (Sawka et al., 2005). Urinary techniques such as urine specific gravity, urine colour and Uosm (with the exception of the freezing point osmometer) are inexpensive tools and provide immediate feedback (Oppliger and Bartok, 2002). For such reasons, these urinary techniques are advantageous and easily mastered by practitioners in sport. Nonetheless, such techniques can result in reduced accuracy and precision in comparison to the "gold standard" tests (Popowski et al., 2001; Oppliger and Bartok, 2002; Shirreffs, 2003).

Urine osmolality is used to measure the number of dissolved particles (osmoles) per unit of water in the urine. For the most accurate measurements of Uosm, a freezing point Osmometer is recommended, a technique that traditionally requires a trained technician (Oppliger and Bartok, 2002). An alternative, inexpensive, method for measuring Uosm is through refractometry, which involves passing a beam of light through a urine sample and measuring how much the beam is refracted.

Although urine analysis is usually an easy and inexpensive method of testing hydration status, it has its drawbacks. Popowski et al. (2001) demonstrated a "lagging" response of Uosm and urine specific gravity behind that of the "gold standard" plasma osmolality changes. His subjects who lost approximately 5 % of their body mass from exercise, then rehydrated by replacing this lost fluid. In these subjects, plasma osmolality increased and decreased in an almost linear fashion but Uosm and specific gravity were found to be less sensitive and demonstrated a delayed response. Therefore, athletes who aggressively

rehydrate following exercise will produce urine that is dilute despite them being dehydrated. Changes in Uosm and colour would mirror the volume of fluids consumed rather than the amount of water retained by the body. These studies suggest that during a state of fluid overload, the kidneys respond by producing large quantities of urine before the body has had time to normalise intracellular and extracellular fluid equilibrium – masking hydration status. Under these circumstances, the validity of urine osmolality is compromised and its use for testing the rehydration of athletes is uncertain. Cheuvront and Sawka (2005) suggested it is best used to assess and distinguish euhydration and dehydration as long as the first void in the morning is used.

To date, there is limited literature reporting normative values of Uosm in elite, senior level football players. Furthermore, whether practical hydration assessment techniques, such as Uosm, are sensitive to changes in training and match load.

2.8.3: Countermovement Jump / Jump testing

The countermovement-jump (CMJ) test is used as an objective, and functional, performance measure. Jump performance is determined by an interaction of several factors, including the muscular coordination of the upper and lower extremity, maximal force developed by the musculature involved, and the rate at which force can be developed (McLellan et al., 2011). The CMJ is test commonly used by many health care professionals, coaches and strength and conditioning professionals to monitor improvements in physical performance (Leard et al., 2007).

The CMJ test is used vastly throughout sport due to its ease of use and low physiological strain on the athlete (Gathercole et al., 2015). Direct measurement of the CMJ kinetic and kinematic variables can provide practitioners with valuable insights into their athletes'

performance status. These insights include: the movement efficiency of the athlete; the athletes' neuromuscular status in response to training and competition; the presence of adaptation or fatigue; and the lower-body explosive qualities of the athlete (Halson and Jeukendrup, 2004; Cormack et al., 2008b; Cormack et al., 2008c; Gathercole et al., 2015). This versatility has enabled the CMJ test in becoming a useful tool in the routine monitoring of athletes in all sporting backgrounds (Nibali et al., 2015).

The most common method of assessing jump performance in sport is via a 'Jump Mat' due to its low cost and portability (Rogan et al., 2015). Leard et al. (2007), reported the 'Just Jump System' (JJS) to provide a valid measure of jump height and a high association ($r = 0.967$, $P < 0.01$) when compared to jump height values derived form a 3-camera motion-capture system. Nonetheless, the validity of jump mats is not as statistically backed in comparison to force platforms, which are considered the reference standard. Rogan et al. (2015) found systematic differences between the two methods; the jump mat resulted in slightly shorter contact times and higher jump heights. In support, McMahon et al. (2016) found that the JJS overestimated CMJ height compared with the force platform (0.46 ± 0.09 m vs. 0.33 ± 0.08 m) but an excellent relationship between CMJ heights were derived from both systems ($r = 0.998$, $P < 0.001$, power = 1.00). Despite research reporting an excellent association between JJS and force platforms (Rogan et al., 2015; McMahon et al., 2016) the large mean difference in jump heights suggests that the JJS does not provide a valid measure of jump height unless using a corrected equation. Force platforms allow for a greater choice of output variables such as flight time: contraction time (FT:CT), peak power (PP- W), and max force (MF - N), average power (AP – W), average force (AF – N), and take-off velocity (TV – m/s). Therefore, CMJ testing using a force platform has shown to provide a more advanced insight into the movement mechanics of a CMJ as well as an overall performance output.

Research in sport has examined the impact of training and match-play on CMJ performance, with reported impairments in CMJ performance immediately after competitive match-play or high intensity exercise, and the subsequent 72-h post-game (Andersson et al., 2008; Leeder et al., 2014; Wiewelhove et al., 2015) and further showed a negative effect on neuromuscular performance (Cormack et al., 2008b). Table 2.3 shows the recovery time course of CMJ indices post football match-play or high intensity activity. The majority of research in football has focused on time course changes immediately following a football match with changes in performance ranging from 0 to 12 % relative to baseline (Andersson et al., 2008; Ispirlidis et al., 2008; Fatouros et al., 2010; Magalhães et al., 2010).

Table 2.3. Summary of studies undertaken on recovery time-course of jump testing protocols in football.

						Mean ± SD	Time point post-activity				
Author	**Participants**	**Playing level**	**Activity**	**Equipment**	**Measurement**	**Pre-activity**	**0 hour**	**Day 1**	**Day 2**	**Day 3**	**Day 4**
Nedelec et al. (2014)	10 professionals	Professional	Football Match	Force platform	CMJ height (cm)	39.9 ± 2.2		36.9 ± 2.9 (-7.5 %)	37.3 ± 3.4 (-6.5 %)	37.4 ± 2.4 (-6.2 %)	
Ispirilidis et al. (2008)	14 elite		Football match (68-min)	Ergojump contact platform	CMJ height (cm)			-9.3 %			
Fatorouros et al. (2010)	20 trained males	U21 1st Division Greece	Football match	Ergojump contact platform	CMJ height (cm)			-10.0 %			
Magalhaes et al. (2010)	16 trained males	2nd/3rd division Portugal	Football match	Bosco's mat	CMJ height (cm)		-12.0 %	-8.0 %	-8.0 %	-8.0 %	
Gathercole et al. (2015)	11 males	College level	3 stage Yo-Yo fatiguing protocol	Force plate	CMJ height (m)	0.44 ± 0.11	0.40 ± 0.09	0.43 ± 0.10		0.46 ± 0.08	
					PP (W/kg)	65.1 ± 7.9	62.8 ± 8.3	63.9 ±7.1		64.7 ± 8.2	
					MF (W/kg)	25.8 ± 4.9	24.5 ± 3.1	25.5 ± 4.3		24.9 ± 2.7	
					FT (s)	0.544 ± 0.045	0.520 ± 0.044	0.534 ± 0.046		0.540 ± 0.050	
					FT:CT	0.753 ± 0.131	0.701 ± 0.123	0.729 ±0.117		0.695 ± 0.106	
Cormack et al. (2008a)	22 ARF	Elite	ARF match	Force plate	FT (s)		-3.6 %	-3.5 %		-0.4 %	-0.1 %
					AP (W)		-9.4 %	-13.0 %		-6.1 %	-1.5 %
					FT:CT		-16.7 %	-17.1 %		-3.7 %	-3.5 %

De Hoyo et al. (2016)	15 young elite	Spanish U19 1st league senior (U19)	Match	Force plate	CMJ height (cm)	36.31 (± 3.83)	-4.2 % (34.80)	-12.9 % (31.64)	-6.2 % (34.11)
					Average Con (N)	1465.18	-2.9%	-3.7%	-4.4%
					Average Ecc (N)	671.29	-3.1%	-2.9%	-0.5%
Russell et al. (2016)	15 Premier League	Premier league (U21)	Match(es) (1-4 games)	Force plate	PP (W)	3628 ± 415		3359 (-269 ± 165)	3518 (-110 ± 151)

Blank cells indicate no data reported; Data presented are means or means percentage (%) from baseline; - Decrease | + Increase |

CMJ –Countermovement jump | FT – Flight time | FT:CT – Flight time: Contraction time | Con – Concentric force | Ecc – Eccentric force | PP – Peak power | AP – Average power | MF – Max force

Ronglan et al. (2006) demonstrated a significant decrease in CMJ height (6.7 ± 1.3 %) over a three-day elite international handball tournament. They also observed physical performance tests over a five-day training camp and jump testing was still 6 % below the baseline test result after a 48-h recovery period on days three and four. These results indicate that the physical loading experienced during elite handball is sufficient to reduce physical performance markers. Similarly, Andersson et al. (2008) analysed the time course of recovery from neuromuscular fatigue (NF) between two female football matches, they demonstrated CMJ height was significantly lower than baseline values at all testing points post-match (-4.4 ± 0.8 %), suggesting that CMJ is a useful measure of assessing physical performance and NF. However, many investigators examining NF, using the CMJ test, report on only a few variables. The analysis is largely focussed on peak/mean values relating to the concentric phase (Cormie et al., 2009), with reports limited to jump height and peak power (Cormack et al., 2008b). This common practice of measuring overall "outcome" of movement is due to an association with athletic performance (Baker et al., 2001; Sleivert and Taingahue, 2004), nonetheless such outcomes provide no insight into the execution and mechanics of the movement (Gathercole et al., 2015). Given the complex nature of NF, research suggests that this approach may provide limited information in relation to adaptation and fatigue from training and competition (Cormie et al., 2009; Nibali et al., 2015; Gathercole et al., 2015). However, in comparison to laboratory methods for the assessment of NF, jump testing has ecological validity due to the multi-joint movement and easy implementation in an elite sport setting.

Recent research has indicated that CMJ concentric outputs from the 'push-off'/propulsion phase of the jump may be less sensitive to fatigue induced changes, and that altered

movement strategy may give a better indication of NF (de Hoyo et al., 2016, Balloch, 2018). It has been suggested that athletes may maintain jump height under conditions of underlying fatigue by increasing the length of the CMJ-total contraction time or CMJ-eccentric phase. De Hoyo et al. (2016) observed a range of CMJ force platform outputs in fifteen U19 elite male football players belonging to a Spanish first league team after 90-min of an official competitive match. The observation of average eccentric force, which would take part in the contraction time component of the jump, showed a decline 30-min post-match (−4.0 ± 6.3 %). This decline was also evident at 24-h post-match but became unclear at 48-h post-match. More recently, Balloch (2018) undertook a compelling review of CMJ research in elite sport and summarised that the counter-phase portion of the test may be more important in identifying NF rather than that of the performance output. Balloch (2018) recommended that a more comprehensive analysis is required taking into account the interaction between movement strategy as well as jump performance for a greater insight into detecting NF. Previous research in ARF is in support of this. Cormack et al. (2008a) demonstrated numerous CMJ variables were substantially lower during post-match analysis compared to pre-match. However, only six of the 18 force-time variables analysed during single and five repetition jumps had declined substantially following the match. In particular, CMJ height merely showed a small decrease from pre- to post-match, demonstrating a lack of sensitivity for jump height to detect changes from a single AFL match (ES -0.32 ± 0.46). Nonetheless, CMJ FT and FT:CT displayed a substantial decrease post-match and 24-h post. In a similar study, Cormack et al., (2008b) showed CMJ FT:CT to have a substantial reduction throughout a season of elite ARF, indicating a competitive season of elite ARF elicits fluctuations in neuromuscular response.

As a whole, many investigations have reported increased or unchanged FT following acutely fatiguing exercise (Rodacki et al., 2002; Cormack et al., 2008a; Cormack et al., 2008b), and observed diminished eccentric function up to 72-h following fatiguing protocols (Gathercole et al., 2015). Markedly, in many of these studies, alterations in CMJ mechanics were more sensitive and/or enduring in response to fatigue than either CMJ outputs or concentric-focused variables (Gathercole et al., 2015). A number of studies conflict with the conventional thinking and rationale for using the CMJ as a test of neuromuscular status, with observations showing improved results in fatigued conditions (Meister et al., 2013; Malone et al., 2015; Thorpe et al., 2015). Thorpe et al. (2015) analysed the relationship between daily training load and a range of potential measures of fatigue in elite football players during an in-season competitive phase. A small positive correlation was observed ($r = 0.23$) between CMJ performance and total high intensity running (THIR), suggesting improved performance with increased THIR distance during training. Physiologically, you would expect a decline in neuromuscular performance after intense exercise. Nonetheless, a phenomenon called priming/post activation potentiation effect may explain this increase in performance from THIR distance. Undoubtedly, the contrasting results from research may somewhat cloud practitioners' views on the use of CMJ height in detecting the presence of NF and tracking the time-course of recovery. Contrariwise, recent research in professional football has continued to observe load and CMJ response. Russels et al. (2015) examined the relationship between match activities and CMJ PP in professional reserve team players, undertaking 60 to 90-min, over 1 to 4 matches. The study revealed that match THIR and selected acceleration and deceleration variables were related to CMJ PP output at 24-h, but not 48-h, following match-play. PP was the only CMJ variable to be examined, perhaps if they had included the observation of CMJ outputs more closely related to movement

strategy, such as FT:CT, they may have observed changes at 48-h post-match due to the complex biphasic recovery process. Furthermore, Rowell et al. (2017) examined changes in CMJ height and CMJ FT:CT at various pre- to post-match time points and their relationship to accelerometer-derived 'Player Load' in 18 elite Australian A-league football players over three consecutive pre-season games. Both CMJ variables demonstrated sensitivity to medium and high load at zero and 18-h post-match, however FT:CT demonstrated a more likely reduction than jump height. Reductions in FT:CT also persisted at later post-match time points. Thus, FT:CT may be more sensitive to acute changes in load than that of jump height, but further research is required to investigate whether alternative measures derived from CMJ are sensitive to changes in training load in elite football players.

Recovery following prolonged fatiguing exercise is complex and considered bi-phasic (Komi, 2000; Byrne et al., 2004; Doussett et al., 2007). Previous research has demonstrated an immediate reduction in CMJ performance post-match (Cormack et al., 2008b; De Hoyo et al., 2016). This reduction has been attributed to metabolic disturbances that impair excitation-contraction coupling and reduce stretch-reflex sensitivity, muscle stiffness, and force production (Avela et al., 1999; Komi, 2000), which is then followed by a transient recovery phase 24-h to 48-h post-exercise. A secondary decline occurs beyond 48-h, likely caused by an inflammatory and remodelling process that produces a reflex response via intramuscular sensory receptors to the CNS that reduces neural drive and the muscles ability to utilise stiffness-related elastic energy (Alvela et al., 1999; Bishop et al., 2008). Considering the bi-phasic nature of recovery, careful consideration should be given to the various time points at which CMJ tests are administered.

The chosen method for analyses could also be a factor in determining the potential for observed effects. Malone et al. (2018) calculated individual 'Z-scores' for analysis between pre-training CMJ height and subsequent training intensity output. A 'Z-score' is the number of standard deviations the response is above or below the mean of the distribution (calculated using the following formula: (individual players score - individual players average)/(individual players standard deviation)). Their analyses revealed that CMJ Z-scores of -1 related to reductions in THIR outputs, explosiveness and acceleration/deceleration outputs during training. Using Z-scores can allow the chosen analyses to account for individual differences in neuromuscular performance.

The variation in study designs, such as level of competition (reserve or senior level players), time tested in season (preseason or in-season competition), timing of testing on the day, and the load variables used for analysis may all account for the controversial findings reported in professional football players on CMJ response to activity. To enhance practitioner's decision making on selecting the most sensitive CMJ variable best to monitor NF, the reliability of CMJ output variables should be taken into consideration. To date, little information exists regarding the reliability of potential fatigue markers in elite athletes (Table 2.4). Thorpe et al. (2015) demonstrated CMJ height to have very good day-to-day reliability (SEM 1.5 cm; CV = 3.8 %) in 35 male professional football players competing in the English Premier League. These results are in agreement with observations in 15 elite ARF players with intraday reliability estimates of 5 %.

Table 2.4. Intraday reliability of CMJ testing (CV %) from male athletes.

Author	Number of Participants	Playing level	Device	Intraday CV %								
				JH	CT	FT	FT:CT	PP	MF	AP	AF	TV
Cormack et al. (2008)	15	Australian Football League (2 years full-time training)	Force plate	5.0		3.3		2.9	2.2	5.5	1.0	
Gathercole et al. (2015)	11	College level team sport	Force plate	4.9		1.1	5.2	2.7*	4.3*	2.8*	3.1*	2.5
Thorpe et al. (2015)	35	English Premier League	Jump mat	3.8								

Blank cells indicate no data reported; *Force and power values were converted to values relative to body mass;

CMJ –countermovement jump | JH – jump height | CT – contraction time | FT – flight time | FT:CT – flight time: contraction time | Con – concentric force | Ecc – eccentric force | PP – peak power | AP – average power | MF – max force | AF – average force | TV – take of velocity

Nibali et al. (2015) revealed that CMJ testing does not need a familiarisation period in athletic populations, irrespective of the competitive level (high school, college, professional), perhaps due to its similarity to athletic movements where athletes already possess the competency in motor-patterns required. Further, Nibali et al. (2015) found that jump height was the only variable to display a typical error or coefficient of variation (CV) less than the smallest worthwhile change. These findings support early work conducted by Cormack et al. (2008a) showing that the error associated with a large number of force-time variables was in excess of the smallest worthwhile change in performance. Gathercole et al. (2015) conducted reliability testing over three consecutive days and stated that the output variables associated with the movement strategy (countermovement) phase of the jump are less reliable (CV > 5 %) than those concentric outputs from the push-off phase. Understanding the varying range in reliability for CMJ outputs is important to detect meaningful changes in performance or fatigue related manifestations in jump strategy. The increased 'typical error' and poor reliability of the output variables related to the 'counter-phase' portion of the CMJ leaves practitioners unclear regarding which kinetic or kinematic variables are sensitive to NF in elite football players.

Given the high reliability of CMJ testing using jump height, its ease of use, and low physiological strain of the protocol, it aids support for the use of jump testing in a sporting environment. However, research on the validity of CMJ testing for measuring fatigue-induced changes in neuromuscular function suggests that concentric outputs from the push-off phase may present limitations in monitoring the recovery-fatigue status of athletes (Gathercole et al., 2015). Perhaps, a more detailed analysis on mechanical outputs such as contraction time or rate of force development may be more sensitive to

fatigue-induced changes, but the increased typical error of these outputs should be considered when detecting meaningful changes in performance.

In summary, practitioners are still unsure as to which CMJ variable is best suited to detect the presence of NF in professional football players. A valid marker of fatigue should be sensitive to both acute and chronic fluctuations in training and match load (Meeusen et al., 2013), but longitudinal investigations in the literature are sparse (Thorpe et al., 2017; Rowell et al., 2018). More research is needed on the observation of CMJ variables related to both movement strategy and performance output in senior level professional football players throughout a competitive season.

2.8.3.1: Rate of Force Development

Rate of force development (RFD) has been an area of increased interest in recent years; such data is easily collected from CMJ testing performed on a force platform/force plate. RFD is the development of maximal force in minimal time (Moir et al., 2005) and is defined as:

'A change in force (F) with time (t)'

Rate of force development is measured in Newtons per second (N/s), as force is routinely measured in Newtons (N) and time in seconds (s). Force plates are considered the "gold standard" in force measurement (Moir et al., 2005). However, such equipment is expensive and not always accessible outside a laboratory setting.

Interest in RFD has grown, perhaps due to its specificity to sports performance (Martínez-Valencia et al., 2015; Wang et al., 2016). Peak force is typically achieved

within around 250 ms, yet sports movements often occur within a much short time frame. For example, the ground contact phase during sprint running lasts 50-120 ms (Nicol et al., 2006; Beneke and Taylor, 2010). NF is regularly examined using isolated actions, in the form of either isometric, concentric or eccentric actions. However, none of these actions are naturally occurring in ground locomotion (Nicol et al., 2006).

RFD provides an insight into eccentric-loading behaviour, a fundamental component of the stretch-shortening cycle (SSC) movement and neuromuscular function (Nicol et al., 2006). The SSC is where the pre-activated muscle is first stretched (eccentric action) and then followed by the shortening (concentric action; Nicol et al., 2006). Athletic performance consists of SSC movements, such as sprinting, jumping and changing direction (Kallerud and Gleeson, 2013). The nature of the braking phase of the SSC is usually very fast, of short duration and controlled simultaneously by reflex and central neural pathways. Thus, the analysis of such a movement is far more complex than observing concentric/eccentric actions alone and is far more superior in specificity to sport.

Fatigue in the SSC is said to immediately decrease neuro-muscular function through efferent neural drive, a stretch reflex sensitivity related reduction in muscle stiffness, metabolic disturbances and impaired excitation-contraction coupling (Avela et al., 1999; Nicol et al., 2006; Gathercole et al., 2015). Such aforementioned changes could reduce mechanical efficiency of the movement and increase the energy expenditure of repeated contractions (Gathercole et al., 2015; Byrne et al., 2004). Balsalobre-Fernández et al. (2015) demonstrated that training loads were inversely related with RFD during a half squat, where there was a 30.2 % decrease in the RFD observed between the beginning and the end of the season in elite middle and long-distance runners. However, there was

no relationship observed between RFD and rate of perceived exertion. Further to this, Gathercole et al. (2015) displayed meaningful correlations between max RFD and athlete wellbeing. These findings, albeit methodological limitations, support the observation of RFD as measure of fatigue or state of recovery.

The torque-time curve may also be an important aspect in the observation of RFD, Peñailillo et al. (2015) demonstrated that RFD between 100 to 200 ms was a more specific and sensitive measure of indirect eccentric exercise-induced muscle damage induced by eccentric cycling than levels of maximum isometric force production. If muscle damage can be detected during and immediately after exercise, this would allow rapid implementation of recovery strategies and/ or tapering of further training load to reduce fatigue levels.

Although RFD has been shown to be highly variable compared to the more common measures from CMJ analyses, it has used as an early and sensitive marker of muscle damage (Peñailillo1 et al., 2015). Researchers suggest that this output may be more sensitive to changes in response to training or fatigue that exceed the typical error (Nibiali et al., 2015). However, the practicality of the use of RFD as an assessment tool to detect muscular fatigue/muscle damage requires further investigation for its use in elite football players.

2.8.4: Other Biomarkers of Fatigue

The recovery-fatigue variables discussed above are a few of the conventional, and practical, markers used in elite sports to monitor the effects of exercise stress on the body. Many other proteins, metabolites, electrolytes and small molecules can serve as biomarkers for athletes. Since there is no single cause of fatigue, there is also no single biomarker for assessing fatigue. A single measurement of a biomarker does not provide enough diagnostic information related to physiological/physical function. Therefore, researchers have agreed that multiple markers should be measured together when attempting to detect over-training and/or fatigue (Lee et al., 2017).

Research in football has observed alterations in the anabolic and catabolic hormonal environments (Kraemer et al., 2004; Handziski et al., 2006), muscle damage markers (Meister et al., 2011; Heisterberg et al., 2013), immunologic (Rebelo et al., 1998) and redox states after periods of competition and high-intensity training (Andersson et al., 2010; Magalhaes et al., 2010). Additional markers of muscle damage, alongside the conventionally measured CK, such as myoglobin may leak into circulation from resultant muscle damage. Myoglobin released is a more of a short-term marker of damage measurable in blood and may provide an insight into overall muscle stress/damage (Lee et al., 2017). Another useful maker to determine muscle status post-exercise is blood urea nitrogen (BUN), which is a product of protein degradation that can indicate overall protein synthesis vs. breakdown. Observing all three markers of muscle damage/stress would provide more detailed information on muscle status in response to training and match-play than CK alone.

Muscle damage post-match signals a local inflammatory response facilitating the repair, regeneration and growth of muscle cells (Tidball, 2005). Cytokines play an import role

in the inflammatory processes being signalled immediately post-match as part of the acute response to exercise stress. The immune signalling molecule interleukin-6 (IL-6) is produced in larger amounts than any other cytokine; it has both pro inflammatory and anti-inflammatory roles and responds to many stimuli acutely and chronically. Evidently, these aforementioned factors have promoted the popularity of IL-6 as a global measure of inflammation, yet it provides little diagnostic information about chronic inflammation during overtraining in an athlete. Therefore, researchers agree that multiple cytokines should be measured together when attempting to detect chronic inflammation in athletes (Lee et al., 2017). Post-exercise, cytokines are elevated immediately but return to baseline values within minutes to hours after exercise cessation (Suzuki et al., 2002). However, like most biomarkers, cytokines display a large range of inter-individual variability. Therefore, it is important to determine if values are elevated outside their norm in response to stress while the absolute resting levels of biomarkers may not change.

Two omega-3 fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), play an important role in recovery and aid to reduce inflammation, muscle soreness, and the perception of pain from exercise (Bloomer et al., 2009). Omega-3 fatty acids may also have an influence on neuromuscular function and thus an influence on performance. In general, blood levels of omega-3 fatty acids reflect their clinical role more so than dietary intake. Moreover, macronutrient metabolism of protein from tissues may occur with an imbalance between dietary protein intake and dietary protein needs. Such a deficiency results in tissue protein breakdown for a source of essential amino acids needed to maintain critical body functions. The help gauge an athlete's protein status/ protein degradation a combination of biomarkers including albumin, BUN, globulin and amino acid analysis should be observed.

The hormone Testosterone (T) is an anabolic in nature, it is required for promoting protein synthesis, production of red blood cells, and glycogen replenishment in turn reducing protein breakdown (Salvador et al., 1995). Decreased levels of T may indicate that training volume is too high. Cortisol (C) works antagonistically to T and is considered an important catabolic hormone. It is a glucocorticoid hormone secreted from the adrenal cortex in response to physical and psychological stress (Urhausen et al., 1995; Dickerson and Kemeny, 2004). The hormones T and C appear to be among the most commonly investigated biochemical markers of training stress and recovery. In response to exercise, T and C vary in opposite directions and may represent an imbalance between anabolic and catabolic hormones. Since T and C are competitive agonists at the receptor level of muscular cells, it is hypothesised that an increase in training load will result in a decrease in the T:C ratio (Elloumi et al., 2003). The relationship between T and C could therefore be useful in assessing the impact of training and competition, especially during periods of elevated training loads (Filaire et al., 2004; Reitjens et al., 2005; Coutts et al., 2007; Moore and Fry, 2007; Cormack et al., 2008c). However, the expected hormonal response of these measures for early detection of overreaching or overtraining in team sport athletes over extended periods is unclear (Cormack et al., 2008c). Results from long term studies have been equivocal (Filaire et al., 2003; Kraemer et al., 2004; Moore and Fry, 2007) indicating no change in T or T:C but an observed rise in C during a football season, while a reduction in T was the only change noted during 15-weeks of an intensive training block. Nevertheless, poor performance outcomes and suboptimal training adaptations have been reported in football players with a low T:C ratio (Kraemer et al., 2004). Additionally, Hoffman et al. (2005) demonstrated elevated C levels during a season of American football although values were within normal ranges, and T:C was lower at the end of the season while T remained at baseline. Hormone response may be specific to individual sports and cyclic across longer periods, indicating a need for sport

specific profiling (Cormack et al., 2008c). Perhaps consideration should be given for other key biomarkers of endocrine response such as dehydroepiandrosterone (DHEA), growth hormone, insulin-like growth factor 1 or luteinizing hormone that can inform practitioners about training adaptations and non-functional over-reaching. DHEA is a major secretory product of the adrenal glands; it is co-released along with cortisol in response to adrenocorticotropic hormone from the pituitary gland (Liu and Papadopoulos, 2007).

It is also important to consider that the differences in hormonal responses are based on the type and duration of exercise performed. Furthermore, because hormone and other biomarker concentrations in the blood are highly variable among individuals, these markers are best assessed by analysing progressive increases/decreases away from individual baseline measures. Lastly, practitioners in elite sport should consider the time-point of biomarker collection to help determine its applicability to clinical practice. As discussed, the restoration of physiological homeostasis, muscle damage and decreases in physical performance usually takes 48 to 72-h in football players, although several of muscle related biomarkers have been normalised 24-h after a match (Marqués-Jiménez, 2017). If fatigue is still present but the biomarker is back to pre-exercise values, then the value of such a biomarker is questionable. It is important that the biomarkers collected provide insight into an athlete's physiological response to exercise stress and allow practitioners to, if indicated, modify subsequent training and/or recovery sessions.

2.9: Determining Clinical Significance

Interpretation of research in elite sport is often difficult due population differences, varying protocols and equipment used. Firstly, population differences present difficulties when trying to apply results of studies to 'elite' athletes. For example, literature can sometimes be misleading and state participants are of an elite level involved in 'first-team' squads but are under 19 years of age (U19 level) and would not be classed as 'senior' elite football players. Responses to exercise stress can present differences in those elite athletes that have only recently achieved first team status and those which have been regularly undertaking league football up to three times per week for several years. Therefore, considering the applicability of the study population to clinical practice is of paramount importance.

Secondly, evidenced-based practitioners need to determine if the observed changes can be classified as a "real" change or simply "error" or "noise" in the test. Statistical significance only addresses a hypothesis about whether or not differences exist, statistically, between groups. The outcome of a test can be statistically significant, but not be clinically significant, and vice-versa. Unfortunately, there are no standards for calculating clinically important changes in outcomes but observing typical error, confidence intervals and effect sizes can help determine the strength and direction of an outcome (Atkinson, 2000; Pyne, 2003; du Prel et al., 2009; Page, 2014). Clinical research is only of value if it is properly interpreted. Aside from P-values, a measure of the effect strength should be reported to help determine clinical significance (Page, 2014).

Understanding 'absolute reliability' of a measurement tool can help predict the magnitude of a 'real' change in individual athletes. The methods used to describe 'absolute reliability' include the standard error of measurements (SEM), coefficient of variation

(CV) and limits of agreement (LOA). Factors that influence reliability include measurement error from any systematic bias (e.g. general learning and/or fatigue effects on the tests) and random error due to mechanical or biological variation (e.g. mental or physical state of the individual between trials; Atkinson and Nevill, 1998). The Typical error of measurement (TEM) or standard error of measurement (SEM) is the within subject standard deviation (Atkinson, 2000), and/or the standard deviation of error of measurement in a test. Its value is the typical error or variation in a subject's value from measurement to measurement. The SEM is commonly calculated from the standard deviation (SD) of differences divided by the square root of 2 (or 0.707 × SD of differences). Atkinson (2000) stated that one SD covers 68 % of the differences, whereas seven-tenths (0.707) of a SD covers about 52 % of differences. This method for calculating typical error follows from the fact that the variance of the difference scores is equal to the sum of the variances representing the typical error(s) in each trail. The statistic is expressed in units of measurement, which is useful since the smaller the SEM the more reliable the measurements. In general, minimal important changes must be beyond the SEM to ensure clinical changes were not due to measurement error (Pyne, 2003; Turner et al., 2015).

Further to calculations of SEM, the coefficient of variation (CV) is a ratio statistic, providing reliability as percentage of consistency (Atkinson and Nevill, 1998). The CV is the ratio of the SD to the mean (average). There are various methods of calculating CV, but the simplest way is shown in the equation below with data from repeated measurements on a single case:

$$CV = 100 * (SD \text{ of sample} / Mean \text{ of sample})$$

The CV statistic, expressed as a percentage, is useful since the reliability of different measurement tools can be compared. A test with poor reliability will be unsuitable for tracking small but meaningful changes in the fatigue status of athletes (Hopkins, 2004). However, practitioners should be aware that the assumption of normality for an assumed 'population' of repeated tests applies to CV in the same aspect as with the SEM (Atkinson and Nevill, 1998). Nonetheless, in-field CV % can be valuable for practitioners interested in the variability an individual athlete's performance between competitions or from field test to field test. Researchers have suggested that the simple method of calculating CV does not reflect adequate reliability for some clinical measures. Calculations that are more appropriate have been described from the mean square error term in a repeated-measures ANOVA model. The described percentile of measurement error (68 or 95 %) should also be stated, but these analyses are less apparent in literature and often not stated in the analyses (Atkinson and Nevill, 1998).

One of the most important indicators of clinical significance is the effect size. It reflects the magnitude of the difference in outcomes between groups, with a greater effect size indicating a larger difference (Page, 2014). The equation below was established by Cohen (1988) for traditional calculation of effect values based on group differences:

$$\textbf{Effect size } (\boldsymbol{d}) = \frac{\text{Mean difference between groups}}{\text{SD of both groups}}$$

Cohen quantified effect sizes (Table 2.5) that have been operationally described in ranges. The effect sizes may be positive or negative thus indicating the direction of the effect (Page, 2014).

Table 2.5. Thresholds for interpreting Cohen's D effect size.

Effect Size	Relative size
< 0.2	Trivial effect
0.2 – 0.6	Small effect
0.6 – 1.2	Moderate effect
1.2 0 2.0	Large effect
> 2.0	Very large effect

To determine the smallest worthwhile changes (SWC) in performance, practitioners in sport have commonly used the pooled SD x 2 (small effect size change), assuming that the data are reliable and free from systematic bias. The SWC can help determine what the smallest change in score that can be accepted as 'real' is. The SWC in test scores should be over and above the SEM for the test (Pyne, 2004; Turner et al., 2015).

Lastly, confidence intervals (CI) are the upper and lower boundaries within which a population parameter is thought to lie. The confidence level of 95 % is usually selected meaning that the CI covers the true value in 95 of 100 studies performed. The advantages of observing CI are that they reflect the results at the level of data measurement, and they can indicate the magnitude and direction of the effect studied, offering greater clinical value. The observation of CIs can also be determined for statistical significance, as such if the confidence interval does not include the value of zero it is classed at statistically significant (du Prel et al., 2009; Page, 2014).

Practitioners in elite sport should pay careful attention to the cohort, protocol, and measurement device used to collect the data that may contribute to the variability of the

measurements. Further, the statistical outputs in research should be carefully interpreted to better utilize the evidence to improve their clinical decision-making that may subsequently influence future practice.

2.10: Summary of Literature Review

Professional football involves stochastic exercise; it is a high intensity sport that requires a large number of explosive, repetitive, and eccentric actions with short periods of recovery. At elite level, the demands of the competitive schedule require players to frequently play matches and train with relatively short recovery durations. Therefore, it is likely that players will suffer from transient, cumulative and residual fatigue following training and competition. Fatigue is an integral part of the training process, without it supercompensation and adaptation would not occur. However, fatigue response needs to be carefully monitored so that maladaptation does not occur. Player monitoring may provide an insight into an individual's recovery from intense training or competitive match-play. Although data exists observing changes in physical performance parameters and recovery-fatigue markers in the hours and days following match-play, the present thesis will attempt to quantify the sensitivity of these commonly collected markers to both acute and longitudinal fluctuations in load in elite senior first team football players. This thesis will investigate, for the first time, markers of fatigue and physical performance tests over a complete football season in elite senior players.

Chapter 3: General Methodology

3.1: Participants

All participants, which took part in these studies, were from a full-time professional football senior team competing in the English Football League (EFL) Championship. During the 2014-15 season a total of 42 players made professional appearances for the club but only 18 met the criteria for the analysis (mean ± SD: age 25.9 ± 9.1 yrs; body mass 74.2 ± 6.8 kg; stature 1.8 ± 0.6 m; body fat % 8.2 ± 1.9 %). During the 2015-16 season a total of 33 players made professional appearances, with 21 being including in subsequent analyses (mean ± SD: age 26.8 ± 4.6 yrs; body mass 79.6 ± 8.3 kg; stature 1.8 ± 0.8 m; body fat % 9.4 ± 1.9 %). Players were removed from the analyses if they did not met the criteria for data collection time points, number of appearances or match-min played during the season. The protocols were fully explained to the participants and any questions were answered before their participation in these studies. Written consent was given from players as part of their contractual agreement with the club. The study was approved by the Ethics Committee of the School of Sport and Biological Sciences, University of Bolton. Inclusion in these studies only took into account the data from fit, healthy players for the analyses. Exclusion criteria included any players that sustained an injury during the data collection windows. Goalkeepers were also excluded from all analyses due to positional differences in training and game demands. Data was collected in-field as part of routine testing and monitoring within the clubs' training facility.

3.2: Design and Procedure

All data were collected as part of routine testing and monitoring from pre-season until the end of the season. All measures were carried out at the same time-of-day (09:30 to 11:00 h) to reduce the potential influence of circadian rhythmicity. Data were collected every one-game-week three days apart: on a Friday 24-h before match-day (MD -1, 24-h pre-match) and on a Monday 48-h post-match day (MD +2, 48-h post-match), either side of a scheduled Saturday fixture. The same procedures were followed during two-game-weeks, when matches took place on a Tuesday/Wednesday and on a Saturday. The experimental sessions required all players to provide a urine sample to assess hydration, followed by a blood sample to assess CK concentration and finally perform a CMJ test. Over the course of the season a limited number of data points met the requirement for analysis in each study. The generic training and competition plan followed by the team was developed by the technical staff. The typical weekly training and match schedule is displayed in Table 3.1.a and 3.1.b dependent on a one or two match-week.

Table 3.1.a. Typical weekly micro-cycle based on a one match-play week.

	MONDAY 1	**TUESDAY 2**	**WEDNESDAY 3**	**THURSDAY 4**	**FRIDAY 5**	**SATURDAY 6**	**SUNDAY 7**
AM	Data Collection Training Focus: Starters = technical / 2nd day recovery Non-starters = technical & SSG	Training Focus: Extensive (big pitch size e.g 11 v 11)	Off	Training Focus: Intensive (SSG, unit work/sub-group & tactical themed drills)	Data Collection Training Focus: Tactical (e.g formation & set-plays)	Off	Off / recovery (Gym based recovery modalities)
PM	Off	Gym Session: Weights (heavy)	Off	Gym Session: Weights (power)	Off	Match Post-match: Substitute conditioning runs	Off

'Off' denotes athletes had no prescribed session or fixture and did not need to attend to the clubs training facilities.
'Recovery Modalities' may have included stationary bike, foam rolling/passive stretching, contrast bathing, cold water immersion, cryotherapy chamber, soft tissue massage, compression garments and supplementation.
SSG = Small sided games

Table 3.1.b. Typical weekly micro-cycle based on a two match-play week.

	MONDAY 1	**TUESDAY 2**	**WEDNESDAY 3**	**THURSDAY 4**	**FRIDAY 5**	**SATURDAY 6**	**SUNDAY 7**
AM	Data Collection Training Focus: Tactical (e.g formation & set-plays)	Off	Off / recovery (Gym based recovery modalities)	Training Focus: Starters = technical / 2nd day recovery Non-starters = technical & SSG	Data Collection Training Focus: Tactical (e.g formation & set-plays)	Off	Off / recovery (Gym based recovery modalities)
PM	Off	Match Post-match: Substitute conditioning runs	Off	Gym Session: Weights	Off	Match Post-match: Substitute conditioning runs	Off

'Off' denotes athletes had no prescribed session or fixture and did not need to attend to the clubs training facilities.
'Recovery Modalities' may have included stationary bike, foam rolling/passive stretching, contrast bathing, cold water immersion, cryotherapy chamber, soft tissue massage, compression garments and supplementation.
SSG = Small sided games

3.3: Recovery-Fatigue Markers

3.3.1: Hydration Testing – Urine Osmolality

Players reported to the training facilities and provided a urine sample, the first urine void of the day if possible, to assess hydration status using a handheld urinary refractometer (Osmocheck pocket pal OSMO, Vitech Scientific Ltd, Japan). The players were asked to provide a sample of midstream morning urine directly into a 30-ml, clear, sterile, plastic container, which were analysed within 30-min of collection to provide urine osmolality (Uosm). Prior to analysis, the device was calibrated according to the manufacturer's instructions, by pipetting a drop of distilled water directly on the face of the prism to calibrate the refractometer to point zero. Between samples the prism was rinsed with distilled water and dried according to the manufacturer's guidelines. The reproducibility for Uosm measured via the Osmocheck has been previously reported (CV % 34; ICC 0.979; TE 0.15; Sparks and Close, 2013).

3.3.2: Creatine Kinase Testing

Upon completion of the hydration test, the players provided a blood sample in a rested state to test plasma CK concentrations. The fingertip was cleaned with 95 % ethanol and dried with cotton wool to remove excess fluid prior to blood collection. Capillary blood (32 μL) samples were collected via a finger prick with a safety lancet (Sarstedt DS1588, Numbrecht, Germany) into a heparinized capillary tube (Reflotron®) while at rest in a sitting position. The finger was not squeezed with excessive force to minimise the chance of haemolysing the specimen or diluting the sample with interstitial fluid. From this, the blood was directly pipetted on a CK test strip (Reflotron®) and subsequently analysed through a Boehringer Mannheim Reflotron Analyzer (Reflotron®). All plasma CK concentrations were analysed immediately in a controlled laboratory setting (20 to 22ºC).

The measurement range for CK using this method was 24.4 to 1400 μL. The reproducibility for CK measured via the reflotron has been previously reported (CV % 18 - 20.0; ICC 0.90; TE 94; Harper et al., 2016; Chrismas et al., 2017).

3.3.3: Physical Performance Testing - Countermovement Jump

After this, all players performed a CMJ test. Prior to testing all players performed a standardised warm-up consisting of a 5-min cycle at 80-W power load (Keiser M3, Fresno, CA, USA), dynamic stretching and three submaximal CMJ efforts. After this, the athletes were asked to perform the test standing on a jump-mat (Just Jump System, Perform Better Limited, Southam, Warwickshire, UK) with hands on hips and were asked to drop down to a self-selected level before jumping maximally. The test was repeated three times with a rest period of 30-s provided in between each jump with the highest value used for further analysis. To minimise the influence of external factors (such as possible ground-surface variations during the season), all testing sessions were conducted at the same indoor gym facility to provide consistent stable flooring. This test has previously been reported to be both valid and reliable (Markovic et al., 2004).

The same procedure was followed for the data collected in the 2015-2016 season, however, the CMJ test was performed using a portable force platform (HUR Labs Force Platform, 3.8.0.2, Kokkola, Finland), sampling at a frequency of 1,200-Hz at 16-bit resolution, according to previously described methods (Oliver et al., 2008). The variables that were collected and assessed are outlined in Table 3.2.

Table 3.2. CMJ force platform output variables collected during testing.

CMJ Variable	Abbreviation	Description	Measurement unit
Height	**H**	The maximum jump height achieved, calculated using take-off velocity	cm
Contraction time	**CT**	The duration from jump initiation to take-off from the force platform	ms
Flight time	**FT**	Time spent in the air from jump take-off to landing	ms
Flight time: contraction time	**FT:CT**	The ratio of flight time to contraction time	
Max force	**MF**	Greatest force achieved during the jump	N
Peak power	**PP**	Greatest power achieved during the jump	W
Average force	**AF**	Mean force generated during the concentric phase of the jump	N
Average power	**AP**	Mean power generated during the concentric phase of the jump	W
Take-off velocity	**TV**	Greatest velocity achieved during the jump	m/s

3.4: Load Monitoring

3.4.1: Global Positioning System

Load monitoring data was collected using a portable 10-Hz GPS device (Catapult system Minimax S4; Sprint 5.1.7, Catapult Sports, Melbourne, Australia) worn inside a custom-made garment positioned between the scapula during every training session over the course of the season. To achieve high signal quality and allow for satellite lock, all GPS devices were activated 15-min before data collection prior to placing the units on the football players. The minimum acceptable number of available satellite signals was 8 (range 8 to 11; Rawstorn et al., 2014). The dwell time (minimum effort duration) was set at 0.2 s to detect all moderate and high intensity efforts; this was the manufacturer recommended and default setting. To avoid inter-unit error all players were told to wear the same GPS device for each training session (Jennings et al., 2010). Data collected were downloaded and exported post-session, and subsequently analysed. This technology enabled us to follow the three-dimensional movement of an individual player to be tracked over time. The GPS devices have been shown to provide valid and reliable estimates of instantaneous velocity during accelerations, decelerations, and constant velocity movements during multidirectional, linear and soccer-specific activities (Coutts and Duffield, 2010; Aughey, 2011; Varley et al., 2012; Rampinini et al., 2015; Bastida et al., 2018). The physical and intensity parameters collected during the training sessions during the 2015-16 season are displayed in Table 3.3.

Table 3.3. GPS training load parameters collected during all training session.

Training Parameter	Description	Measurement unit
Training time	Training minutes from the session duration	min
Total distance (TD)	Oedometer - Distance covered in all speed zones	m
Total high intensity Distance (THID)	Vel zone 5 - sprint + high speed running	m
Total sprint distance (TSD)	Vel zone 4 – distance covered above 7 m/s	m
High speed running (HSR)	Vel zone 3 – distance covered between 5.5 to 7 m/s	m
High intensity accelerations (H.I Accels)	Accel zone 8: speeding up > 4 m/s^2	m/s^2
High intensity decelerations (H.I Decels)	Accel zone 1: slowing down > -4 m/s^2	m/s^2
Moderate intensity accelerations (M.IAccels)	Accel Zone 6: speeding up between 1 to 2.49 m/s^2	m/s^2
Moderate intensity decelerations (M.I Decels)	Accel Zone 3: slowing down between -2.49 to -1 m/s^2	m/s^2
Player load (PL)	Arbitrary unit calculated from accelerations, decelerations and vertical force	Arbitrary unit (AU)
Player load/ minute	Player load ÷ session duration	PL/min
Metres/ minute	Total distance ÷ session duration	M/min

3.4.2: Match External Load – Prozone®

Matches were analysed using a semi-automated multi-camera recognition system (Prozone® 3, 10.0.0, Leeds, England). The Prozone® system allowed the tracking of all players simultaneously during all home league matches and only the away league matches where the system was in situ. These cameras capture the players' movements individually during matches and are based upon the development of a continuous movement trajectory. This method has previously been found to be reliable for the measurement of match activity in football, with data demonstrating a CV of 16.2 ± 6.4 % and 95 % CI of 15.6 to 16.7 (Gregson et al., 2010). The data was analysed using proprietary software to create a dataset on each player's physical performance data. Physical performance variables selected for the analysis were: (1) total distance covered (TD); (2) total high-intensity running distance (THID – distance covered above 5.5 m/s); (3) total sprint distance (TSD – distance covered over 7 m/s); (4) total high-intensity run number (HIN – number of high-intensity efforts above 5.5 m/s); (5) total sprint number (TSN – number of high-intensity efforts above 7 m/s); (6) total number of explosive sprints (EXS – number of efforts when accelerating at high-speed into a sprint and remaining in the high-speed run threshold, 5.5 to 7 m/s, for a period of less than one second); (7) total number of medium-acceleration (MAcc – speeding up at 2.5 to 4 m/s^2); (8) total number of high-accelerations (HAcc – speeding up at ≥ 4 m/s^2); (9) total number of medium-decelerations (MDec – slowing down at -2.5 to -4 m/s^2); and (10) total number of high-decelerations (HDec – slowing down $\geq$ -4 m/s^2).

Chapter 4: Effects of a 90-min football match on recovery-fatigue markers and physical performance testing in elite English Football League Championship players.

4.1: Introduction

Fatigue is a multifactorial, complex phenomenon, which is difficult to define and measure. Match-related fatigue can be described as a decline in physical performance during the hours and days' post-match leading to levels of sub-optimal physiological function (Rampinini et al., 2011). Therefore, in elite football, players are monitored to provide objective measures on their physiological response to training and games and gain an insight into their general fatigue and recovery status. To make informed decisions on player status it is important that tests are sensitive enough to detect meaningful changes in physiological status or physical performance (refer to Chapter 2.9 for determining clinical significance). In order to provide an insight into fatigue, several measures have been used such as CK concentrations, CMJ testing and hydration levels. The validity of these fatigue markers is relatively unknown in football populations (Thorpe et al., 2015; de Hoyo et al., 2016).

The collection of CK, via capillary blood, is minimally invasive and provides immediate feedback, making it an advantageous marker of recovery. The CMJ test has been used due to ease of use and low physiological strain on the athlete and can be used an indicator of neuromuscular performance (Gathercole et al., 2015). Research conducted in football supports the observation of CK and CMJ outputs for detecting acute and residual fatigue post-match or post high-intensity activity (Nedelec et al., 2014; de Hoyo et al., 2016), but little to no research has been conducted on elite players after a 90-min competitive game (Scott et al., 2016).

Lastly, monitoring player hydration status has become a key component in sport performance due to the negative impact of dehydration on exercise performance (Cheuvront et al., 2003). Recommendations of total body water measurements and

plasma osmolality are the current “gold standard” assessments but are impractical in football due to the analytical expertise and methodological control required (Armstrong et al., 1994; Cheuvront and Sawka, 2005). As a result, urine osmolality (Uosm), measured via a handheld refractometer, has been used as a reliable measure of acute body water loss (Oppliger et al., 2005), but little research has been conducted on post-match changes in elite players.

Considering the differences in genetic makeup and training history of elite footballers and lower-level players or sedentary individuals, responses to competition/training stress and recovery over time may differ (Barnett, 2006). A comprehensive body of research has investigated post-match acute and residual fatigue responses to match-play, but differences between subject status and study design call into question the ecological validity and relevance to football.

Therefore, the purpose of this research was to examine the acute changes from competitive match-play in physiological status and physical performance as part of routine testing and monitoring of senior first-team players over the course of a professional football season.

4.2: Methods

Participants

Eighteen first-team professional male football players were recruited for this study which took place during the 2014-15 season as described in Chapter 3 (section 3.1). Exclusion criteria included any players that sustained an injury during the data collection windows.

Experimental Design

All data were collected as described in Chapter 3 (section 3.2). For the purpose of this Chapter, only the data of players who had completed 90-min or more of competitive match-play between MD -1 and MD +2 were used for subsequent analysis. Over the course of the season a limited number of data points met the requirement for analysis (Hydration, n = 44; CK, n = 14; CMJ Height, n = 48).

Hydration Testing – Urine Osmolality

Hydration testing was measured as described in Chapter 3 (section 3.3.1).

Creatine Kinase Testing

Creatine Kinase testing was measured as described in Chapter 3 (section 3.3.2).

Physical Performance Testing - Counter-movement Jump testing

The CMJ (a vertical jump test) was performed using a jump-mat (Just Jump System, Perform Better Limited, Southam, Warwickshire, UK) as described in Chapter 3 (section 3.3.3).

Statistical Analysis

The data were analysed by means of the Statistical Package for Social Sciences (SPSS) for Windows (SPSS, Chicago, IL), IBM version 26, using paired t-tests for all variables pre- and post-match. The normality of distribution was checked using the Shapiro-Wilk test. Effect sizes (ES) were calculated from the ratio of the mean difference to the pooled standard deviation. The magnitude of the ES was classified as trivial (≤ 0.2), small (> 0.2–0.6), moderate (> 0.6–1.2), large (> 1.2–2.0) and very large (> 2.0) based on the guidelines from Batterham and Hopkins (2006) as described in Chapter 2 (section 2.9). The results are presented as the mean ± SD throughout the text unless otherwise stated. Following convention, the alpha level of significance was set at 5 %, hence values where $P < 0.05$ have been referred to as ‘‘significant’’ and those where $P < 0.10$ but > 0.05 as a ‘trend’’ or ‘‘marginally significant’’. When the SPSS output demonstrated significance levels of $P = 0.000$, these were corrected to $P < 0.0005$ (Kinear and Gray, 1995).

4.3: Results

Table 4.1 shows the group mean ± SD for Uosm, CK concentration and CMJ height pre- and post-match, along with informative statistics for interpreting clinical and meaningful changes. Figure 4.1 illustrates the changes in physical performance and recovery-fatigue markers.

CK concentrations were significantly higher 48-h post 90-min of competitive football compared to 24-h pre-match (t_{13} = -3.409, P = 0.005, ES = 0.89, 41 %). Jump height decreased pre to post-match with a statistically significant mean change of 2.1 cm (95 % CI, 0.5 to 3.8), t_{47} = 2.578, P = 0.013, ES = -0.33, -3.9%. There was no change in Uosm pre- to post-match (P > 0.05).

The positional changes pre to post-match in Uosm, CK and CMJ height are shown in Table 4.2. Full-backs displayed the greatest increase in CK 24-h pre to 48-h post-match with a very large effect size change (+62.5 %, ES: 0.205). Attackers demonstrated the largest percentage decrements in CMJ height, followed by full-backs (7.7 and 4.7 %, respectively). Central midfielders, wide midfielders and attackers all displayed similar increases in Uosm pre to post-match after 90-mins of competitive football.

Figures 4.2.a – c. display the average individual changes in Uosm, CK and CMJ height 24-h pre to 48-h post-match after 90-mins of competitive football.

Table 4.1. Mean (± SD) values for hydration (Uosm), CK concentrations and CMJ height pre-match (MD -1) and post-match (MD +2).

	Pre-Match	Post-match	Mean Difference	% Diff	Std. Error Mean	Effect Size	CI (95 %)	P-value
Hydration (mOsmols)	260 ± 88	286 ± 117	26 ± 122	10.1 ↑	18.4	0.35	-63 to 11	0.163
CK (u/l)	264 ± 97	372 ± 142	108 ± 119	41.0 ↑	31.8	0.89	40 to 177	**0.005**
CMJ Height (cm)	54.5 ± 6.7	52.4 ± 6.1	-2.1 ± 5.7	-3.9 ↓	0.8	-0.33	0.5 to 3.8	**0.013**

Statistical significance ($P < 0.05$) is indicated in **bold**. The magnitude of the ES was classified as trivial (≤ 0.2), small (> 0.2–0.6), moderate (> 0.6–1.2), large (> 1.2–2.0) and very large (> 2.0). ↑ denotes positive % change, while ↓ denotes negative % change.

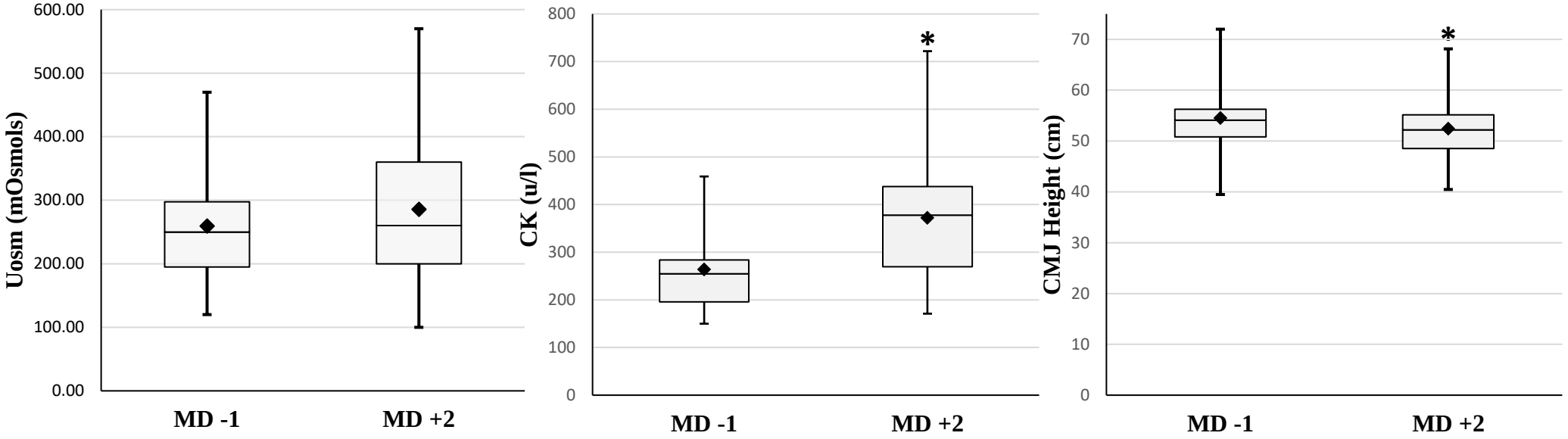


Figure 4.1. Box and whisper plots illustrating changes in Uosm (mOsmols), CK concentration (u/l) and CMJ height (cm) 24-h pre and 48-h post 90-min of competitive football.

◆*Indicates average.*

***Indicates significant change.*

Table 4.2. Positional differences (mean ± SD) for hydration (Uosm), CK concentrations and CMJ height pre-match (MD -1) and post-match (MD +2).

	Pre-match (MD -1)	Post-match (MD +2)	% difference	ES change
Hydration (mOsmols)	**260 ± 88**	**286 ± 117**	**10.1 ↑**	**0.25**
Central Defenders (n=2)	*238 ± 59*	*246 ± 88*	*3.3 ↑*	*0.10*
Full-backs (n=4)	*268 ± 94*	*279 ± 107*	*4.1 ↑*	*0.11*
Central Midfielders (n=5)	*268 ± 95*	*305 ± 125*	*13.9 ↑*	*0.34*
Wide Midfielders (n=4)	*252 ± 106*	*289 ± 112*	*14.8 ↑*	*0.34*
Attackers (n=3)	*293 ± 85*	*343 ± 235*	*17.0 ↑*	*0.28*
CK (u/l)	**264 ± 97**	**372 ± 142**	**41.0 ↑**	**0.89**
Central Defenders	*213 ± 89*	*327 ± 89*	*53.5 ↑*	*1.28*
Full-backs	*255 ± 68*	*415 ± 86*	*62.5 ↑*	*2.05*
Central Midfielders	*370 ± 153*	*438 ± 276*	*18.4 ↑*	*0.30*
Wide Midfielders	*228 ± 46*	*317 ± 101*	*39.0 ↑*	*1.14*
Attackers	-	-	-	-
CMJ Height (cm)	**54.5 ± 6.7**	**52.4 ± 6.1**	**-3.9 ↓**	**-0.33**
Central Defenders	*51.9 ± 5.6*	*50.8 ± 3.7*	*-2.2 ↓*	*-0.24*
Full-backs	*58.5 ± 10.4*	*55.6 ± 8.8*	*-4.7 ↓*	*-0.28*
Central Midfielders	*54.4 ± 2.9*	*52.8 ± 5.2*	*-2.9 ↓*	*-0.38*
Wide Midfielders	*52.6 ± 3.3*	*50.4 ± 3.9*	*-4.2 ↓*	*-0.61*
Attackers	*56.6 ± 2.0*	*52.2 ± 6.6*	*-7.7 ↓*	*-0.88*

The magnitude of the ES was classified as trivial (≤ 0.2), small (> 0.2–0.6), moderate (> 0.6–1.2), large (> 1.2–2.0) and very large (> 2.0). ↑ denotes positive % change, while ↓ denotes negative % change

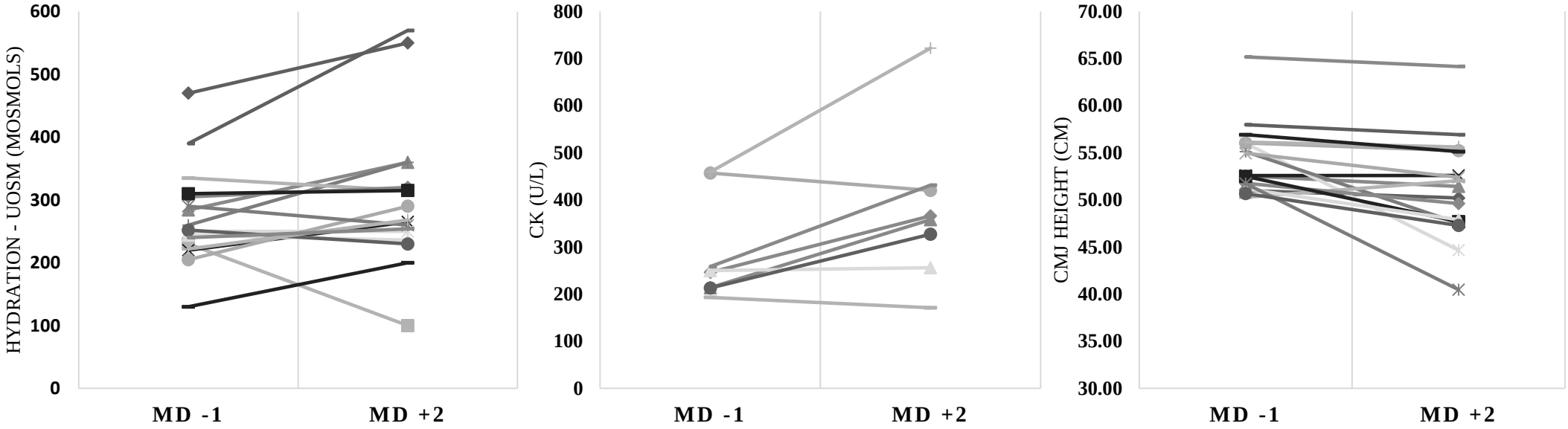


Figure 4.2.a – c. Individual changes 24-h pre to 48-h post 90-min of competitive football in Uosm (mOsmols), CK concentration (u/l) and CMJ height (cm).

Shapes indicate individual average.

4.4: Discussion

The main finding of this study was that CK concentrations are significantly increased and physical performance (CMJ) is significantly impaired 48-h post-match in comparison to the 24-h pre-match baselines. Throughout a competitive football season, a congested schedule causes a quick turnaround in fixtures with limited recovery time to restore normal homeostasis. Therefore, monitoring for spikes in CK concentrations and alterations in physical performance may provide clinically relevant insights of individual players prior to the next training session and/or fixture, equipping practitioners with advantageous knowledge concerning the recovery-fatigue status.

The pre- to post-match change in CMJ height of -2.1 cm (-3.9 %, P = 0.013) is lower in comparison to studies of a similar nature which report greater impairments in physical performance 24 to 48-h post-match (Magalhaes et al., 2010; Nedelec et al., 2014; de Hoyo et al., 2016). The inconsistency in findings may be attributable to differences in testing procedures, equipment used, the time-point of data collection, and population differences. Research conducted by de Hoyo et al. (2016) reported a decline in CMJ height of 6.1 % from pre to 24-h post-match, whereas Nedelec et al. (2014) reported a 7.5 % decline. It must be noted that both these declines were observed at 24-h post-match and are therefore not directly comparable with the findings of the present study due to the bi-phasic nature of recovery. At 48-h post-match, research in football has reported declines ranging from 6.5 % to 8.0 % (Magalhaes et al., 2010; Nedelec et al., 2014). However, the use of testing equipment is not standardised in research and studies have mainly used 'Just Jump Systems'/'Jump Mats' and force platforms which display large mean calculated differences in jump height as described in Chapter 2 (section 2.8.3). It has been established that systematic differences are present between the jump mat and the force platform, with the jump mat resulting in slightly shorter contact times and

higher jump heights, unless a corrected equation is used (Rogan et al., 2015, McMahon et al., 2016). This explains the substantial variability in CMJ height reported in the literature in football. To add to the inconsistency in findings, two studies undertaken in elite football found positive associations between CMJ height and THIR completed during training (Malone et al., 2015; Thorpe et al., 2015). These findings add controversy to the original rationale of using CMJ height as a measure of NF. Finally, previous studies assessing CMJ performance in elite football has had small sample sizes (Ispiridis et al., 2008; Malone et al., 2015; de Hoyo et al., 2016) compared to our moderate sample size of 48 data points, which meets set criteria over the course of the season, and explains some of the differences compared to current studies in the literature.

To determine if the pre- to post-match change in CMJ height is classified as a meaningful change in performance practitioners should take into consideration the typical error and CV. A study performed by Nibali et al. (2015) found CMJ height to be the only variable to display a percent CV (3.5 % CV) and typical error (0.01 m) less than their calculated SWC. The SWC for jump height was stated as 0.02 m (2 cm or 4.9 %), suggesting that CMJ would need to decline by at least 2 cm for it to be classified as a meaningful change in performance. In the present study, a change in CMJ height of 2.1 cm is outside of the standard error of mean (0.8 cm) and outside the typical error reported by Nibali et al. (2015). Additionally, the mean change pre- to post-test of 3.9 % in the present study is outside the CV % previously reported but has not met the SWC of 4.9 % (Nibali et al., 2015). Collectively, this information suggests that although the CMJ height test was able to detect significant alternations in physical performance after 90-min of elite competitive football, it was questionable whether the test was able to establish a true meaningful change in performance. Despite this, a small ES change and narrow CIs were reported for CMJ height 48-h post-match (ES: -0.33, CI = 0.5 to 3.8 cm), which provides

a better method for assessing real-world change in jump performance. The clinical significance of a 2.1 cm impairment in CMJ height could ultimately affect the outcome of a competitive match. Such a small change can be the difference in out-pacing or out-manoeuvring an opposing player that may yield a competitive performance advantage. It is well established in research that indices of strength and power have been highlighted as predictors of both linear and multidirectional sprint performance (Young et al., 2002; Barr and Nolte, 2011). According to Northeast et al. (2017), relative peak power output from a CMJ predicted linear sprint performance in elite English Premier League football players over 5, 10 and 20 m distances. Therefore, considering this, the clinical significance of a 2.1 cm difference in physical performance is undisputable and relevant.

The present findings indicate that CMJ height does appear to be a useful marker 48-h post competitive match-play in the present population. However, recent literature has suggested CMJ height can be maintained under conditions of underlying fatigue through alterations in the 'counter-movement' phase of the jump (de Hoyo et al., 2016; Balloch 2018). Future research should consider the observation of CMJ outputs related to changes in jump strategy and not overall output to provide further insight into NF post-competition.

CK is reported within the literature as an indicator or EIMD and exercise severity (Owen et al., 2015). The elevated CK concentrations 48-h post-match (372 ± 142 u/l) were somewhat expected and similar to ranges previously reported in similar investigations in elite football (358 ± 176 u/l; de Hoyo et al., 2016). Other research using fingertip capillary blood samples, found CK concentrations 48-h post-match between 467 to 520 u/l in elite football players (Nedelec et al., 2014; Russell et al., 2016; Scott et al., 2016), which varies from our current findings. Differences in post-match means may be due to

differences in the methodological approach and the specific sub-group of elite senior level football players recruited. The current investigation examined pre- to post-match changes across multiple matches over the season in senior players, whereas previous research focuses on changes from one competitive match and/or in athletes < 21 years of age (Ispirlidis et al., 2008; Coelho et al., 2011; Nedelec et al., 2014; de Hoyo et al., 2016). In addition, the homogenous population used in this study, elite senior football players, may have influenced the findings. This population group is more resilient to EIMD compared to younger or novice/recreational athletes. It has been found that skeletal muscle adjusts rapidly to repetitive periods of exercise, such that the muscle is protected against subsequent periods of exercise-induced micro-trauma (McBride et al., 1995), and resulting in lower levels of CK concentration in the blood.

Several researchers have shown associations between CK concentration and high intensity activities completed (McLellan et al., 2011; Young et al., 2012; de Hoyo et al., 2016). Conversely, research by Scott et al. (2016) did not identify any significant associations between physical match performance and CK concentration 48-h post-match in elite senior football players. However, in agreement with the current findings, they did demonstrate a marked difference in CK concentration pre- to post-match. A study with such high external validity applicable to our own suggests that CK may be useful on a more general level of athlete monitoring as it is acutely sensitive to the overall demands of match-play but is not specifically related to locomotor activities completed by elite football players 48-h post-match. The nature of elite first team football and its sport specific demands may impact the physiological resilience of the population used in this study and that observed by Scott et al. (2016), reasoning for the lack of association with match demands and a somewhat low concentration of CK 48-h post competitive match-play.

Effective monitoring strategies in football require the tracking of variables that are sensitive to the physiological changes that accompany the stress of football specific exercise. Previous research has suggested that CK can be used as a marker for the early detection of player fatigue or muscle overload in football players (Lazarium et al., 2009; Coelho et al., 2011). However, a better understanding is required regarding what change in CK concentration is clinically significant considering the large reference range of 82-1083 u/l currently reported in the literature (Mougios, 2007). Therefore, taking these values into consideration, the observed mean after 90-min of elite competitive football is relatively low and would not suggest any significant muscular or fatigue related stress in the players used in this study. Nevertheless, our results still suggest that CK concentrations, collected via capillary blood, are sensitive to the overall demands of match-play.

Hydration measurements revealed no statistical difference between values observed 24-h pre-match (260 ± 88 mOsmols) compared to 48-h post-match (286 ± 117 mOsmols). Post-game hydration scores of 286 ± 117 mOsmols were on the lower end of the measurement reference range (0 to 1500 mOsmols) suggesting that 90-min of competitive elite level football did not induce dehydration 48-h post-game. This testing time point post-match gives sufficient time to allow players to rehydrate. Research has suggested it takes approximately six-h to achieve full rehydration post-match induced dehydration (~2 % of body mass; Shirreffs et al., 1996*)*. This may explain the lack of statistical and meaningful change 48-h post-match. Dehydration greater than 2 % body mass should be avoided as this is the threshold after which performance and cognitive function has shown to be reduced (Cheuvront et al., 2003; Coyle, 2004; Lieberman, 2007; Sawka et al., 2007). A hydration deficit of 2 % body-mass, commonly seen in football (Edwards et al., 2007; Owen et al., 2013), is consistent with a Uosm score of over 900

mOsmols (Maso et al., 2004) and has been correlated with a reduction in physical performance in football matches (Mohr et al., 2010).

Uosm is recognised as a reliable measure of acute body water loss (Armstrong et al., 1994; Oppliger et al., 2005; Kilding et al., 2009; Tam et al., 2011). However, timing is critical when testing Uosm and, in general, the first void of the morning is recommended to minimise confounding influences (such as diet) and maximise measurement reliability (Armstrong et al., 1994; Shirreffs and Maughan, 1998). The present study did not control for these factors. Cheuvront and Sawka (2005) suggested Uosm is best used to assess and distinguish euhydration and dehydration. Therefore, it may not be accurate enough to detect hydrations status in first team professional football players' 48-h post-match.

Lastly, the positional changes 24-h pre to 48-h post-match showed the position of full-back had the greatest increase in CK concentrations (+62.5 %, ES: 0.205). This position has shown to cover greater distance when sprinting than central defenders, central midfielders and attackers (Bradley et al., 2009), and therefore may reason higher concentrations of CK due to the high-velocity mechanics of sprinting. Further, the largest percentage decrement and ES change in CMJ performance was observed in Attackers. This position, alongside central defenders, are known to cover the least distances at lower intensities and high speeds (Bradley et al., 2009). Perhaps this decrement was influenced by other performance factors such as jumping to header the ball that linear physical performance variables do not explain. Repeated vertical force production to win aerial balls may subsequently have a negative effect on CMJ performance post-match.

4.5: Conclusion

After 90-min of competitive elite level football, CMJ height was able to detect an alteration in physical performance in the direction expected. However, the question is raised whether the impairment was a meaningful change in performance and/or of clinical significance. This highlights the importance when it comes to choosing the analysis technique and reporting more than just statistical significance (P-value). Further, the substantial variability in research questions the JJS for its accurate calculation of jump height. Creatine kinase testing seems to be sensitive to the overall demands of match-play associated with 90-min of competitive football but findings do not suggest any significant muscular or fatigue related stress when used in senior elite football players. Hydration measured via Uosm using a handheld urinary refractometer (osmocheck) reflects that elite level football players are able to achieve rehydration by 48-h post-game.

Chapter 5: The impact of a 90-min competitive football match on CMJ, CK and Uosm in elite English Football League Championship players.

5.1: Introduction

This chapter will mirror that of Chapter 4 instead using a more advanced assessment technique for physical performance. Chapter 4 demonstrated that CMJ height, measured on a 'Just Jump System (JJS)' is able to detect physical performance decrements 48-h post 90-min of elite competitive football. Nevertheless, studies have shown that the JJS overestimates CMJ height and a corrected equation should be used in order to provide a valid measure of jump height. Recent research has therefore focused on the "gold standard" of CMJ testing using a force platform, due to its greater precision and sensitivity when appropriately calibrated (Rogan et al., 2015; McMahon et al., 2016).

Research has suggested that concentric outputs from the push-off phase of the CMJ, such as jump height, does not have the resolution to detect signs of NF (Cormack et al., 2008b, de Hoyo et al., 2016, Balloch, 2018). It has been suggested that the CMJ can be performed in fatigued conditions without a deterioration in performance through the modification of movement strategies and/or skill (Knicker et al., 2011). Force platforms allow a greater choice of output variables (peak and average force and power outputs, velocity, rate of force development) from the 'counter-phase' portion of the jump that directly relates to the SSC action to be observed. Therefore, the inclusion of the additional variables from both the push-off and countermovement portion of a CMJ should provide greater specificity to detecting NF in elite athletes.

Considering the limited attention received in the literature in relation to valid markers of fatigue that are sensitive to variability in preceding match/training load (Thorpe et al., 2015; de Hoyo et al., 2016), the collection of Uosm and CK remained the same to remain cohesive throughout the thesis. Therefore, the aims of this research were to: (1) examine the acute changes from competitive match-play in CMJ, CK and Uosm as part of routine

testing and monitoring of senior first-team players over the course of a professional football season; and (2) examine the association between the acute changes in CMJ, CK and Uosm and match external load after 90-min of elite competitive football.

5.2: Methods

Participants

Eighteen first-team professional male football players were recruited for this study, which took place during the 2015-16 season as described in Chapter 3 (section 3.1). Exclusion criteria included any players that sustained an injury during the data collection windows.

Experimental Design

All data were collected as described in Chapter 3 (section 3.2). For the purpose of this study, data were only included from weeks where only one (Saturday) fixture was scheduled. Only players who had completed 90-min or more of competitive match-play between MD -1 and MD +2 were used for subsequent analysis. Over the course of the season a limited number of data points met the requirement for analysis (Hydration, n = 23; CK, n = 35; CMJ, n = 34).

Hydration Testing – Urine Osmolality

Hydration testing was measured as described in Chapter 3 (section 3.3.1).

Creatine Kinase Testing

Creatine Kinase testing was measured as described in Chapter 3 (section 3.3.2).

Physical Performance Testing – Counter-movement Jump

The CMJ (a vertical jump test) was performed using a portable force platform (HUR Labs Force Platform 3.8.0.2, Kokkola, Finland) as described in Chapter 3 (section 3.3.3)

Match External Load – Prozone®

Match external load was analysed using a semi-automated multi-camera recognition system (Prozone®, Leeds, England) as described in Chapter 3 (section 3.4.2).

Statistical Analysis

The data were analysed by means of the Statistical Package for Social Sciences (SPSS) for Windows (SPSS, Chicago, IL), IBM version 26, using paired t-tests for all variables pre- and post-match. The normality of distribution was checked using the Shapiro-Wilk test. Effect sizes (ES) were calculated from the ratio of the mean difference to the pooled standard deviation. The magnitude of the ES was classified as trivial (≤ 0.2), small (> 0.2–0.6), moderate (> 0.6–1.2), large (> 1.2–2.0) and very large (> 2.0) based on guidelines from Batterham and Hopkins (2006) described in Chapter 2 (section 2.9). For correlations, the Pearson method was used to analyse the association between each participants' change in CMJ, CK and Uosm measures pre- to post-match and the individual's match external load, The criteria adopted to categorise the magnitude of the correlation (r) between the different measures were classified as trivial (≤ 0.1), small (> 0.1–0.3), moderate (> 0.3–0.5), large (> 0.5–0.7), very large (> 0.7–0.9), and almost perfect (> 0.9–1.0) based on the guidelines of Hopkins et al. (2009). The results are presented as the mean ± SD throughout the text unless otherwise stated. Following convention, the alpha level of significance was set at 5 %, hence values where $P < 0.05$ have been referred to as ''significant'' and those where $P < 0.10$ but > 0.05 as a 'trend'' or ''marginally significant''. When the SPSS output demonstrated significance levels of $P = 0.000$, these were corrected to $P < 0.0005$ (Kinear and Gray, 1995).

5.3: Results

Table 5.1 shows the group means ± SD for CMJ, CK and Uosm pre- and post-match. Creatine kinase concentrations were significantly higher 48-h post 90-min of competitive football compared to 24-h pre-match (+48.7 %; t_{34} = 3.92, P = 0.001, ES = 0.66). The CMJ output measure AP displayed a moderate effect size change 24-h pre- to 48-h post-match (-7.3 %; t_{33} =-3.67, P = 0.001, ES = -0.63). Small effect size changes were observed in CMJ FT, FT:CT, TV and jump height 24-h pre- to 48-h post-match. Following a 90-min of competitive football, CMJ FT was 13.55 ms lower compared to pre-match (-2.4 %; t_{33} = -2.61, P = 0.014, ES = -0.45). A significant reduction was also observed in FT:CT (-7.4 %; t_{33} = -2.30, P = 0.028, ES = -0.39), TV (-3.3 %; t_{33} = 2.50, P = 0.018, ES = -0.43) and jump height (-4.2 %; t_{33} = -2.02, P = 0.51, ES = 0.35) with lower values 48-h post-match. There was no significant change in CMJ CT, PP, MF and AF, or Uosm pre- and post-match (ES = 0.03 to 0.28; P > 0.05).

Table 5.1. Mean (± SD) values for hydration (Uosm), CK concentrations and CMJ outputs pre-match (MD -1) and post-match (MD +2).

Variable	Pre-Match	Post-match	% Diff	Std. Error Mean	Effect Size	CI (95 %)	P-value
Hydration (mOsmols)	343 ± 201	283 ± 173	-17.6 ↓	53.8	-0.23	-172 to 51	0.273
CK (u/l)	436 ± 272	648 ± 407	48.7 ↑	54.1	0.66	102 to 322	**0.001**
CMJ Height (cm)	36.3 ± 5.7	34.8 ± 5.0	-4.2 ↓	0.8	-0.35	-3.1 to 0.01	*0.051*
CMJ Contraction Time (ms)	859.3 ± 108.8	878.3 ± 105.2	2.2 ↑	11.5	0.28	-4.5 to 42.4	0.110
CMJ Flight Time (ms)	562.4 ± 31.7	548.8 ± 38.5	-2.4 ↓	5.2	-0.45	-24.1 to -3.0	**0.014**
CMJ FT:CT	0.69 ± 0.16	0.64 ± 0.11	-7.4 ↓	0.02	-0.39	-0.1 to -0.01	**0.028**
CMJ Peak Power (W)	4323.4 ± 897.5	4130.6 ± 515.3	-4.5 ↓	126.7	-0.26	-450.6 to 64.9	0.138
CMJ Max Force (N)	1934.8 ± 374.5	1875.9 ± 176.1	-3.0 ↓	64.4	-0.16	-190.0 to 72.2	0.367
CMJ Take-off Velocity (m/s)	2.69 ± 0.30	2.60 ± 0.18	-3.3 ↓	0.03	-0.43	-0.15 to -0.02	**0.018**
CMJ Average Power (W)	1119.3 ± 148.7	1037.5 ± 187.9	-7.3 ↓	22.3	-0.63	-127.2 to -36.4	**0.001**
CMJ Average Force (N)	1058.1 ± 58.8	1056.5 ± 72.1	0.2 ↓	9.5	0.03	-21.0 to 17.8	0.868

Statistical significance ($P < 0.05$) is indicated in **bold**, and a trend (where $P < 0.10$ but > 0.05) is indicated in *italics*. The magnitude of the ES was classified as trivial (≤0.2), small (>0.2–0.6), moderate (>0.6–1.2), large (>1.2–2.0) and very large (>2.0). ↑ denotes positive % change, while ↓ denotes negative % change.

Table 5.2 shows the mean ± SD of the positional match averages for the physical match performance parameters collected through the Prozone® system, where players covered total distances of 10.2 ± 0.8 km on average during each match. Players performed more MAcc (108 ± 18) and HAcc (15 ± 6) compared to MDec (86 ± 18) and HDec (8 ± 4) on average each game, respectively. A total of 120 high-intensity runs above 5.5 m/s (HIN) were performed, while 38 of these runs were high-intensity efforts above 7 m/s (TSN). Central midfielders covered a greater total distance than wide midfielders, full-backs, attackers and central defenders (Table 5.2). Central and wide midfielders also covered a greater distance in high-intensity running and sprint distance than full-backs, central defenders and attackers.

Table 5.2. Mean (± SD) values for match external load variables collected through the Prozone® system during matches.

External Load Variable	Central Defenders (n = 18)	Full-Backs (n = 15)	Central Midfielders (n = 13)	Wide Midfielders (n = 6)	Attackers (n = 8)	Match average
Total distance covered (m)	9656 ± 484	10399 ± 831	11014 ± 783	10084 ± 438	9727 ± 150	**10222 ± 805**
Total high intensity running distance > 5.5 m/s (m)	632 ± 130	973 ± 259	1154 ± 240	1089 ± 203	945 ± 236	**940 ± 283**
Total sprint distance >7 m/s (m)	171 ± 49	284 ± 116	324 ± 105	344 ± 133	276 ± 143	**271 ± 120**
Number of high-intensity efforts (>5.5 m/s)	84 ± 16	126 ±30	151 ± 27	124 ± 14	119 ± 17	**120 ± 32**
Number of high-intensity efforts (>7 m/s)	22 ± 6	42 ± 15	49 ± 14	44 ± 15	37 ± 16	**38 ± 16**
Number of explosive sprints (5.5 to 7 m/s)	8 ± 2	16 ± 7	21 ± 8	15 ± 5	15 ± 5	**15 ± 7**
Total number of medium accelerations (2.5 to 4 m/s^2)	87 ± 8	111 ± 13	121 ± 14	120 ± 15	109 ± 16	**108 ± 18**
Total number of high accelerations (>4 m/s^2)	11 ± 3	15 ± 5	15 ± 6	18 ± 4	17 ± 7	**15 ± 5**
Total number of medium decelerations (-2.5 to -4 m/s^2)	66 ± 9	89 ± 15	101 ± 19	85 ± 11	88 ± 14	**85 ± 18**
Total number of high decelerations (> -4 m/s^2)	6 ± 3	10 ± 3	9 ± 4	8 ± 5	9 ± 5	**8 ± 4**

Associations between the measured variables are shown in Table 5.3. Important findings were: there were moderate associations between changes in CMJ FT and AP to HID, HIN, EXS and MAcc ($r = -0.395$ to -0.496, $P < 0.05$). Pre- to post-match changes in CMJ FT also displayed moderate associations to TSD ($r = -0.395$, $P = 0.034$), TSN ($r = -0.446$, $P = 0.015$) and MDec ($r = -0.423$, $P = 0.022$). Increases in CMJ CT were associated with HIN ($r = 0.39$), and CMJ AF with HIN, EXS and medium accelerations/decelerations ($r = -0.397$ to 0.459) completed during the match. Urine osmolality demonstrated a strong association to the number of HAcc ($r = 0.561$, $P = 0.019$). No significant associations were observed between changes in CMJ height, FT:CT, PP, MF and CK to Prozone® match external load.

The positional pre to post-match changes in Uosm, CK and CMJ are shown in Table 5.4.a – c. Central midfielders displayed the greatest ES change in Uosm, CK, CMJ height, and CMJ FT in the direction expected. Wide and central midfielders demonstrated the greatest ES changes in CK concentrations 24-h pre to 48-h post-match (ES: 0.81 -0.93). Further, central midfielders and full-backs displayed the greatest changes in CMJ FT:CT, TV and AP after 90-min of competitive football. Central defenders displayed the greatest percentage change for decrements in CMJ peak power and max force outputs (6.6 – 8.9 %), however wide midfielders displayed the greatest ES change in these outputs (ES: 1.01 – 1.06).

Figures 5.1.a – k display the average individual changes in Uosm, CK and CMJ 24-h pre to 48-h post-match after 90-mins of competitive football.

Table 5.3. Correlations (r) and significance between match external load and Uosm, CK and CMJ (± denote direction of r; * donates statistical significance $P < 0.05$)

Variable	TD (m)	HID (m)	TSD (m)	HIN	TSN	EXS	MAcc	HAcc	MDec	HDec
Hydration (mOsmols)	r = 0.002	r = 0.125	r = -0.044	r = 0.380	r = 0.085	r = 0.430	r = 0.358	r = 0.561*	r = 0.348	r = 0.033
CK (u/l)	r = -0.047	r = 0.086	r = 0.079	r = 0.111	r = 0.136	r = 0.273	r = 0.103	r = 0.313	r = 0.106	r = -0.011
CMJ Height (cm)	r = -0.159	r = -0.108	r = -0.077	r = -0.186	r = -0.108	r = -0.124	r = -0.060	r = -0.031	r = -0.068	r = -0.107
CMJ Contraction Time (ms)	r = 0.277	r = 0.304	r = 0.187	r = 0.388*	r = 0.239	r = 0.388	r = 0.258	r = 0.104	r = 0.236	r = 0.092
CMJ Flight Time (ms)	r = -0.321	r = -0.419*	r = -0.395*	r = -0.451*	r = -0.446*	r = -0.496*	r = -0.438*	r = -0.294	r = -0.423*	r = -0.369
CMJ FT:CT	r = -0.252	r = -0.211	r = -0.116	r = -0.273	r = -0.118	r = -0.217	r = -0.076	r = -0.006	r = -0.222	r = -0.303
CMJ Peak Power (W)	r = -0.094	r = -0.025	r = 0.032	r = -0.65	r = 0.067	r = 0.038	r = 0.130	r = 0.089	r = -0.016	r = -0.216
CMJ Max Force (N)	r = -0.056	r = -0.027	r = 0.030	r = -0.042	r = 0.069	r = 0.034	r = 0.111	r = 0.081	r = -0.060	r = -0.204
CMJ Take-off Velocity (m/s)	r = -0.12	r = 0.42	r = -0.18	r = 0.03	r = 0.038	r = 0.066	r = 0.132	r = -0.049	r = 0.178	r = 0.040
CMJ Average Power (W)	r = -0.384*	r = -0.391*	r = -0.312	r = -0.487*	r = -0.365	r = -0.398*	r = -0.380*	r = -0.194	r = -0.358	r = -0.202
CMJ Average Force (N)	r = -0.281	r = -0.356	r = -0.261	r = -0.422*	r = -0.356	r = -0.459*	r = -0.448*	r = -0.141	r = -0.397*	r = -0.202

Table 5.4.a. Positional differences (mean ± SD) for hydration (Uosm), CK concentrations and CMJ pre-match (MD -1) and post-match (MD +2).

	Pre-match (MD -1)	Post-match (MD +2)	% difference	ES change
Hydration (mOsmols)	**343 ± 201**	**283 ± 173**	**-17.6**	**-0.23**
Central Defenders	*220 ± 44*	*210 ± 50*	*-4.5*	*-0.21*
Full-backs	*469 ± 267*	*283 ± 175*	*-39.7*	*-0.83*
Central Midfielders	*237 ± 133*	*440 ± 376*	*85.9*	*0.72*
Wide Midfielders	*263 ± 47*	*267 ± 60*	*1.3*	*0.06*
Attackers	*330 ± 156*	*248 ± 98*	*-24.7*	*-0.63*
CK (u/l)	**436 ± 272**	**648 ± 407**	**48.7**	**0.66**
Central Defenders	*447 ± 317*	*711 ± 530*	*58.9*	*0.60*
Full-backs	*491 ± 291*	*704 ± 393*	*43.3*	*0.62*
Central Midfielders	*408 ± 230*	*642 ± 339*	*57.2*	*0.81*
Wide Midfielders	*326 ± 130*	*453 ± 141*	*38.8*	*0.93*
Attackers	*409 ± 367*	*400 ± 206*	*-2.0*	*-0.03*
CMJ Height (cm)	**36.3 ± 5.7**	**34.8 ± 5.0**	**-4.2**	**-0.35**
Central Defenders	*38.9 ± 7.0*	*39.0 ± 5.2*	*0.3*	*0.02*
Full-backs	*38.8 ± 5.6*	*35.8 ± 4.0*	*-7.5*	*-0.60*
Central Midfielders	*33.0 ± 2.4*	*30.6 ± 1.5*	*-7.3*	*-1.20*
Wide Midfielders	*35.6 ± 3.4*	*34.2 ± 3.1*	*-3.9*	*-0.42*
Attackers	*31.4 ± 2.4*	*30.1 ± 1.2*	*-4.2*	*-0.69*

The magnitude of the ES was classified as trivial (≤ 0.2), small (> 0.2–0.6), moderate (> 0.6–1.2), large (> 1.2–2.0) and very large (> 2.0). ↑ denotes positive % change, while ↓ denotes negative % change

Table 5.4.b Positional differences (mean ± SD) for CMJ CT, FT and FT:CT pre-match (MD -1) and post-match (MD +2).

	Pre-match (MD -1)	Post-match (MD +2)	% difference	ES change
CMJ CT	**859.3 ± 108.8**	**878.3 ± 105.2**	**2.2**	**0.28**
Central Defenders	*888.4 ± 86.0*	*882.5 ± 91.9*	*-0.7*	*0.07*
Full-backs	*827.3 ± 88.6*	*882.3 ± 112.9*	*6.6*	*0.54*
Central Midfielders	*834.2 ± 62.4*	*851.7 ± 79.9*	*2.1*	*0.24*
Wide Midfielders	*754.8 ± 158.6*	*792.5 ± 153.3*	*5.0*	*0.24*
Attackers	*972.7 ± 96.1*	*963.0 ± 61.3*	*-1.0*	*-0.12*
CMJ FT	**562.4 ± 31.7**	**548.8 ± 38.5**	**-2.4**	**-0.45**
Central Defenders	*577.5 ± 28.1*	*580.7 ± 31.3*	*0.6*	*0.11*
Full-backs	*576.2 ± 31.6*	*547. 9 ± 40.2*	*-4.9*	*-0.78*
Central Midfielders	*554.5 ± 7.5*	*531.7 ± 20.3*	*-3.9*	*-1.42*
Wide Midfielders	*557.9 ± 25.6*	*551.3 ± 30.4*	*-1.2*	*-0.24*
Attackers	*521.3 ± 26.67*	*505.2 ± 13.1*	*-3.1*	*-0.77*
CMJ FT:CT	**0.69 ± 0.16**	**0.64 ± 0.11**	**-7.3**	**-0.39**
Central Defenders	*0.72 ± 0.24*	*0.67 ± 0.09*	*-8.2*	*-0.32*
Full-backs	*0.70 ± 0.05*	*0.63 ± 0.11*	*-9.8*	*-0.83*
Central Midfielders	*0.67 ± 0.05*	*0.63 ± 0.07*	*-5.5*	*-0.60*
Wide Midfielders	*0.77 ± 0.23*	*0.72 ± 0.19*	*-6.7*	*-0.25*
Attackers	*0.54 ± 0.07*	*0.53 ± 0.03*	*-2.8*	*-0.28*

The magnitude of the ES was classified as trivial (≤ 0.2), small (> 0.2–0.6), moderate (> 0.6–1.2), large (> 1.2–2.0) and very large (> 2.0). ↑ denotes positive % change, while ↓ denotes negative % change

Table 5.4.c. Positional differences (mean ± SD) for CMJ PP, MF and TV pre-match (MD -1) and post-match (MD +2).

	Pre-match (MD -1)	Post-match (MD +2)	% difference	ES change
CMJ PP	**4323.4 ± 897.5**	**4130.6 ± 515.5**	**-4.5**	**-0.26**
Central Defenders	*5064.7 ± 1359.3*	*4614.9 ± 660.0*	*-8.9*	*-0.42*
Full-backs	*4063.8 ± 285.6*	*3946.3 ± 217.6*	*-2.9*	*-0.46*
Central Midfielders	*4136.4 ± 459.1*	*4061.3 ± 391.6*	*-2.8*	*-0.18*
Wide Midfielders	*4014.0 ± 72.2*	*3859.5 ± 204.0*	*-3.8*	*-1.01*
Attackers	*3779.9 ± 118.3*	*3793.8 ± 86.9*	*0.4*	*0.13*
CMJ MF	**1934.8 ± 374.5**	**1875.9 ± 176.1**	**-3.0**	**-0.16**
Central Defenders	*2144.4 ± 636.5*	*2003.4 ± 195.2*	*-6.6*	*-0.30*
Full-backs	*1843.2 ± 146.7*	*1794.2 ± 128.4*	*-2.7*	*-0.36*
Central Midfielders	*1910.0 ± 66.1*	*1953.5 ± 166.4*	*2.3*	*0.34*
Wide Midfielders	*1916.4 ± 120.0*	*1819.2 ± 48.0*	*-5.1*	*-1.06*
Attackers	*1725.1 ± 33.9*	*1720.2 ± 49.5*	*-0.3*	*-0.12*
CMJ TV	**2.69 ± 0.30**	**2.60 ± 0.18**	**-3.1**	**-0.43**
Central Defenders	*2.84 ± 0.46*	*2.75 ± 0.18*	*-3.2*	*-0.26*
Full-backs	*2.75 ± 0.20*	*2.65 ± 0.15*	*-3.8*	*-0.59*
Central Midfielders	*2.55 ± 0.09*	*2.45 ± 0.06*	*-3.7*	*-1.2*
Wide Midfielders	*2.64 ± 0.13*	*2.59 ± 0.12*	*-1.9*	*-0.41*
Attackers	*2.48 ± 0.10*	*2.43 ± 005*	*-2.0*	*-0.67*

The magnitude of the ES was classified as trivial (≤ 0.2), small (> 0.2–0.6), moderate (> 0.6–1.2), large (> 1.2–2.0) and very large (> 2.0). ↑ denotes positive % change, while ↓ denotes negative % change

Table 5.4.d Positional differences (mean ± SD) for CMJ AP and AF pre-match (MD -1) and post-match (MD +2).

	Pre-match (MD -1)	Post-match (MD +2)	% difference	ES change
CMJ AP	**1119.3 ± 148.7**	**1037.5 ± 187.9**	**-7.3**	**-0.63**
Central Defenders	*1222.0 ± 105.9*	*1201.6 ± 124.4*	*-1.7*	*-0.18*
Full-backs	*1159.7 ± 135.9*	*984.3 ± 231.5*	*-15.1*	*-0.92*
Central Midfielders	*968.1 ± 78.6*	*875.3 ± 111.7*	*-9.6*	*-0.96*
Wide Midfielders	*1153.8 ± 100.2*	*1085.9 ± 74.4*	*-5.9*	*-0.77*
Attackers	*994.8 ± 141.9*	*961.0 ± 81.6*	*-3.4*	*-0.29*
CMJ AF	**1058.1 ± 58.8**	**1056.5 ± 72.1**	**-0.2**	**0.03**
Central Defenders	*1094.3 ± 83.5*	*1124.5 ± 69.1*	*2.8*	*0.39*
Full-backs	*1036.2 ± 30.6*	*1005.7 ± 60.2*	*-2.9*	*-0.64*
Central Midfielders	*1039.8 ± 47.4*	*1034.1 ± 43.1*	*-0.5*	*-0.12*
Wide Midfielders	*1049.1 ± 55.8*	*1030.4 ± 61.9*	*-1.8*	*-0.32*
Attackers	*1054.3 ± 29.0*	*1059.9 ± 30.0*	*0.5*	*0.19*

The magnitude of the ES was classified as trivial (≤ 0.2), small (> 0.2–0.6), moderate (> 0.6–1.2), large (> 1.2–2.0) and very large (> 2.0). ↑ denotes positive % change, while ↓ denotes negative % change

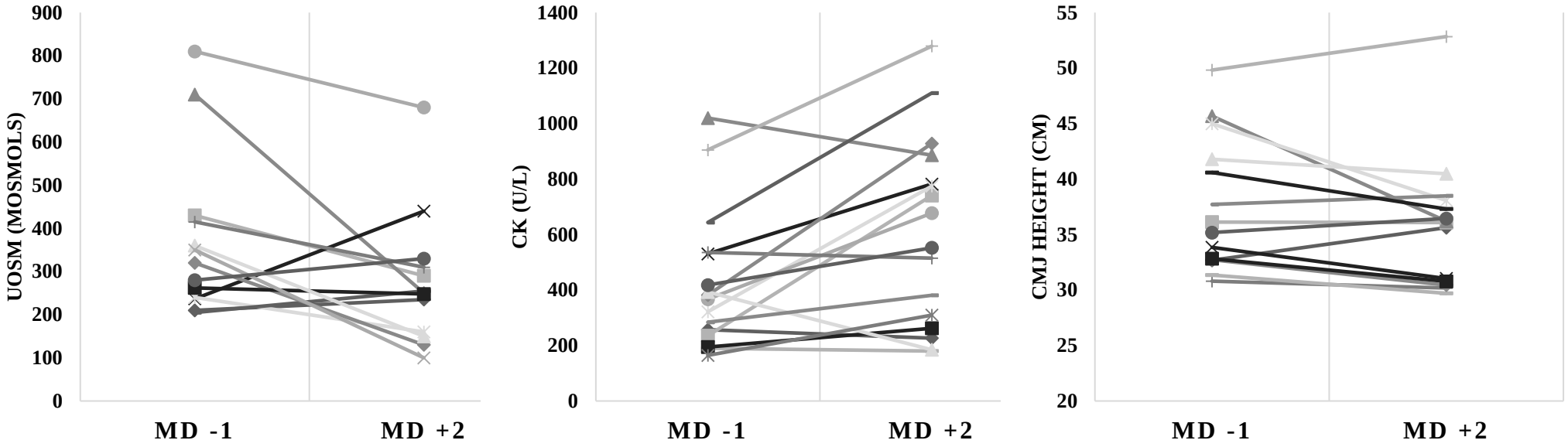


Figure 5.1.a – c. Individual changes 24-h pre to 48-h post 90-min of competitive football in Uosm (mOsmols), CK concentration (u/l) and CMJ height (cm).

Shapes indicate individual average.

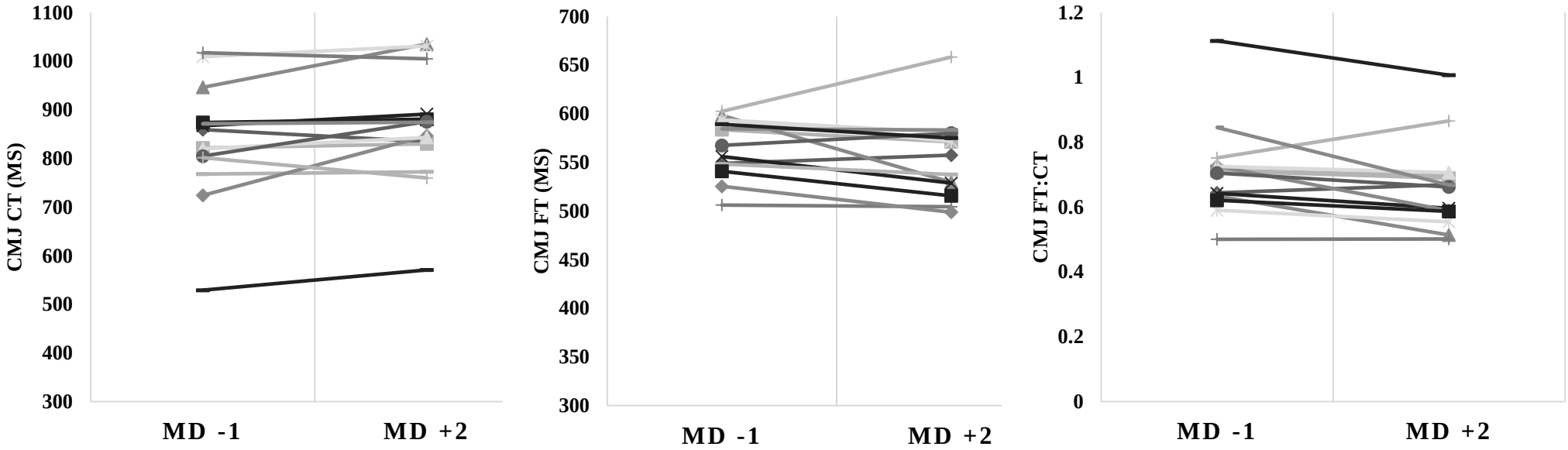


Figure 5.1.d – f. Individual changes 24-h pre to 48-h post 90-min of competitive football in CMJ CT (ms), FT (ms) and FT:CT.

Shapes indicate individual average.

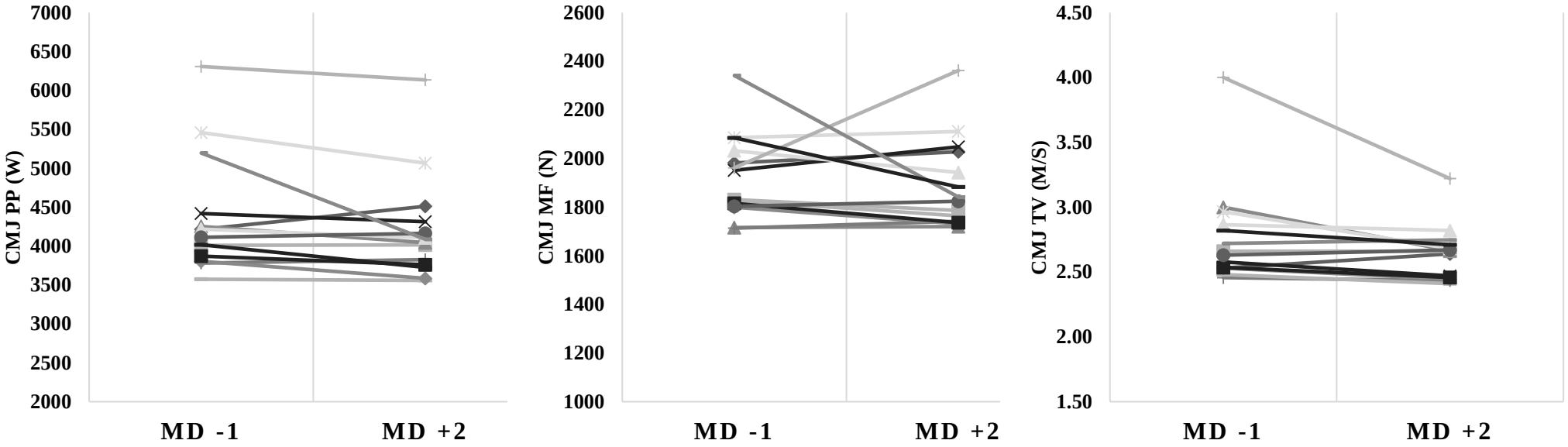


Figure 5.1.g – i. Individual changes 24-h pre to 48-h post 90-min of competitive football in CMJ PP (W), MF (N) and TV (m/s)

Shapes indicate individual average.

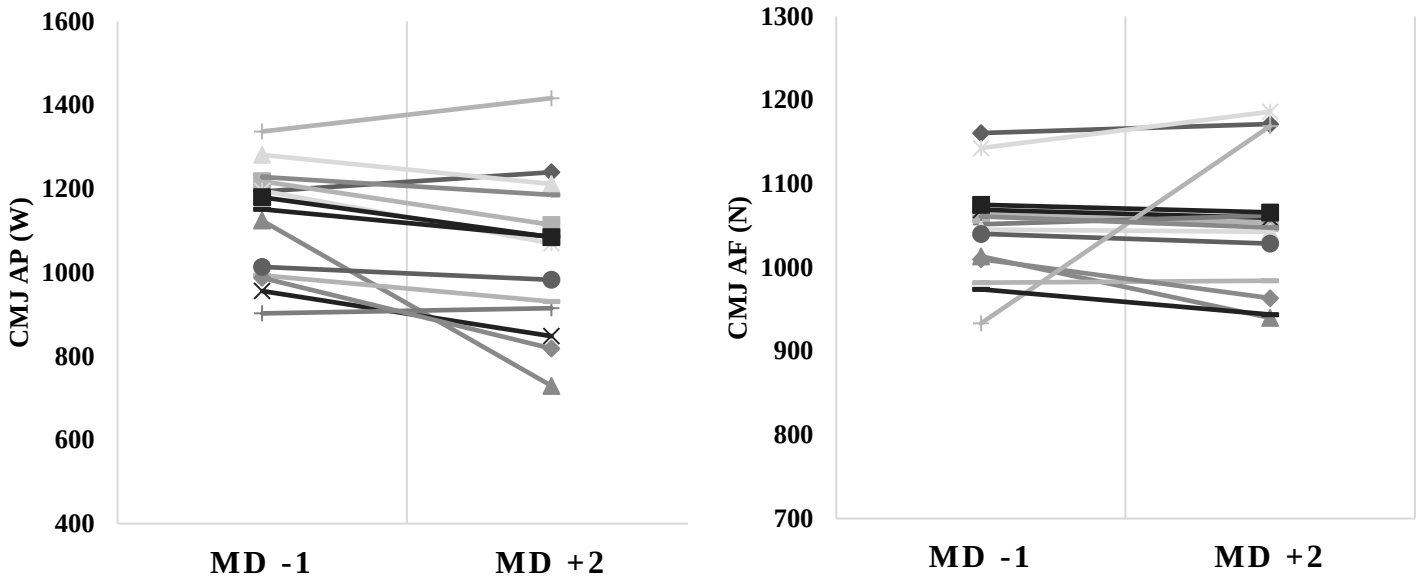


Figure 5.1.j – k. Individual changes 24-h pre to 48-h post 90-min of competitive football in CMJ AP (W) ans AF (N).

Shapes indicate individual average.

5.4: Discussion

The main finding of the study was that CK concentrations significantly increased and CMJ performance measures significantly decreased 48-h post-match compared to 24-h pre-match. These results, which were collected as part of a routine testing and monitoring system over the course of a season, provide a valuable insight into real-world changes during the 48-h recovery window. These findings highlight the challenges faced with competing at a high level, such as recovering to train effectively and potentially being able to play again a few days later.

The findings of the present investigation are in agreement with previous studies (Cormack et al., 2008b; McLean et al., 2010; de Hoyo et al., 2016), but research conducted in professional first-team competitive football remains sparse (Nedelec et al., 2014; Scott et al., 2016). The magnitude of change in CK observed following 90-min of competitive football were as expected, showing a significant increase (212 ± 320 u/l, P = 0.001) and a moderate ES change (ES 0.66). The post-match CK levels are comparable with previously reported research in football, which observed values of 520 and 950 u/l 48-h post-match (Ispirlidis et al., 2008; Scott et al., 2016). However, in relation to the reported reference range in elite level football players (64.9 to 1971.7 u/l), the post-match CK concentrations are low and would suggest minimal muscular or fatigue related stress, or good recovery was present at this time-point of data collection (Mahmutyazicioglu et al., 2018).

The pre-match CK concentrations in the present study were higher compared to previously reported values 24-h pre-match (436 *vs.* 248 to 290 u/l: Nedelec et al., 2014; de Hoyo et al., 2016). Data from training weeks when only one Saturday fixture was

scheduled was included in our study, therefore the elevated pre-match concentrations cannot be associated to that of a mid-week fixture and is therefore purely associated to the training stimulus. In addition, our results also reveal a large variation in CK concentrations, as previously noted in literature (Lazarim et al., 2009; Coelho et al., 2011; Mahmutyazicioglu, 2018). The prospect of cumulative fatigue was considered as data was collected from across a whole season, potentially adding to the variance observed. To account for large variability in CK response, some researchers have defined individuals as either responders or non-responders (Newham et al., 1986; Clarkson et al., 1992; Chen, 2006; Brancaccio et al., 2010). The individual changes pre to post-match, displayed in figure 5.1.a – k, demonstrate how particular individuals are more responsive to exercise stress. Certain individuals present minimal changes in CK concentration, whereas in contrast others display large responses post 90-mins of competitive football. However, the exact underlying reasons for such individual responses are relatively unknown (Heled et al., 2007).

Further, our study observed pre- to post-match changes in CMJ FT, AP and FT:CT, which demonstrated the most sensitivity and magnitude of change. Research surrounding outputs from the 'push-off' phase of the CMJ have previously reported reductions in CMJ height lasting up to 48-h post-game (Magalhes et al., 2010; De Hoyo et al., 2016). Our findings did not find any statistically significant difference between pre and post-game measures for CMJ height, which may be explained by a smaller percentage change in scores (4.2 *vs*. 6.2 to 7.5 %) compared to other studies (Nedelec et al., 2014; de Hoyo et al., 2016). Despite this, the pre- to post-match decrease in CMJ height (1.5 cm) was outside the SE of mean (0.8 cm), displayed a small effect size change and narrow CIs 48-h post-match (ES: -0.35, CI = -3.1 to 0.01 cm), which provides a better method for assessing real-world change in jump performance. In support of concentric outputs from

the push-off phase, AP demonstrated a moderate change (ES: -0.63) and a significant reduction in performance (P = 0.014). This reduction has been attributed to a reduced central drive and/or metabolic disturbances that impair excitation-contraction coupling and reduce stretch-reflex sensitivity, muscle stiffness and force production (Avela et al., 1999; Komi, 2000; Tofari et al., 2018). This is then followed by a transient recovery phase 24-h to 48-h post-exercise. A secondary decline occurs beyond 48-h, likely caused by an inflammatory and remodelling process that produces a reflex response - via intramuscular sensory receptors to the central nervous system (CNS) - that reduces neural drive and the muscles ability to utilise stiffness-related elastic energy (Alvela et al., 1999; Bishop et al., 2008). This inflammatory response is supported by the present study through the observation of significant changes in CK concentrations at 48-h post-match. Careful consideration should be given to the various time points at which CMJ tests are administered, that may partially explain why at 48-h post-match there were substantial impairments in CMJ height, CT, or peak power/force outputs.

The present study provides evidence for the observation of concentric outputs from the push-off phase of the jump. Research suggests that concentric outputs lack sensitivity and that an altered movement strategy may provide a better indication of NF (Cormack et al., 2008a; de Hoyo et al., 2016; Balloch, 2018). A study conducted by de Hoyo et al. (2016) analysed average eccentric force, which accounts for part of the total CT, following 90-min of a competitive football match. Average eccentric force declined 30-min and 24-h post-match but became unclear at 48-h. There was a small increase in CT 48-h post-competition (ES: 0.28, P = 0.110), and a significant reduction in AP (ES: -0.63, P = 0.009). This may reflect low frequency fatigue manifested in an altered movement strategy whereby countermovement range and speed are changed in order to achieve peak values (Andersson et al., 2008; Cormack et al., 2008a). This may have

greater implications than first expected, as the time taken to perform a football specific action can influence success. Furthermore, de Hoyo et al. (2016), demonstrated reduced CMJ height at all time points suggesting that both concentric outputs from the push-off phase and movement strategy outputs are sensitive to fatigue-induced changes from 90-min of competitive football. The results from the current study support this theory, with FT:CT remaining reduced 48-h post-competition (ES: -0.39, P = 0.028). Similar results have been documented following a competitive match in Australian Rules football (Cormack et al., 2008b). The ratio of FT:CT may prove to be an effective marker for detecting NF due to its reflection of outputs concerning concentric push-off (FT) and altered movement strategy (CT). This allows practitioners to identify athletes who are unable to produce concentric force and/or adjust their movement strategy.

Hydration measured via Uosm did not reveal any statistically significant difference pre to post-match. The post-game hydration scores were low and below the accepted euhydration cut-off set at < 700 mOsmols (Cheuvront and Sawka, 2005). These findings are consistent with those from Chapter 4, suggesting that 90-min of competitive football at the elite level does not induce dehydration 48-h post-match, and/or hydration strategies during and post-match are sufficient to maintain euhydration.

In relation to the second aim of the study, limited associations were observed between pre- to post-match changes in CMJ, CK and Uosm with match external load. The pre- to post-match change in Uosm demonstrated a strong statistically significant positive association to the number of HAcc in the present study, but the clinical significance of this association due to reasons discussed previously are negligible. The current study was also unable to identify statistically significant associations between match external load and CK concentration. Our study is in agreement with the findings of Scott et al. (2016)

who were unable to identify statistically significant correlations between Prozone® physical match performance data and CK concentration 48-h post-match in Premier League football players. However, a marked difference in CK concentration pre- to post-match was observed, which is similar to our findings, showing that 90-min of professional match play leads to significant increases in CK concentrations (Nedelec et al., 2014; de Hoyo et al., 2016; Scott et al., 2016; Hecksteden and Meyer, 2020). Nonetheless, a limitation of the study by Scott et al. (2016) was the exclusion of acceleration and deceleration data, and their relationship to CK concentrations. Decelerations require intense eccentric contractions of various muscle groups (Young et al., 2012), and are known to induce cell membrane muscle damage as described in Chapter 2 (section 2.4; Nosaka and Newton, 2002). It could be suggested that due to the superior athletic status of professional footballers, having an increased tolerance to load and their muscles are able to cope with the higher demands placed upon them without reaching 'breaking point'. If regular starters are undertaking 90-min of competitive football up to three-times in seven-days, these players may physiologically adapt better to accommodate this load. Although lacking statistical significance, the present study displayed a small to moderate positive associations between pre- to post-match CK and the number of MAcc, MDec and HAcc ($r = 0.103$ to 0.313). Whilst de Hoyo et al. (2016) reported moderate correlations between GPS distance covered, accelerations, and decelerations, to changes in CK concentrations in elite footballers from one competitive match, their results should be interpreted with caution due to their small sample size ($n = 7$) whom competed in a single 90-min match. Data from other team sports have demonstrated that physical variables, such as impacts and tackles, are more highly associated with CK than that of movement patterns. This would support the notion that CK is influenced by superficial damage associated with injuries. Nonetheless, in one of the few studies where ultra-structural muscle damage (measured via muscle biopsies)

and CK were measured, conducted by Fielding et al. (1993), it was found that no correlation was present between z-band damage and CK activity. It is plausible that the efflux of intracellular proteins, such as CK, results from both structural damage and transient changes in membrane permeability (Brancaccio et al., 2007). Collectively, these findings suggest that CK testing is sensitive to the overall demands of match-play but should not be used to quantify the magnitude of muscle damage in football. In order to gain full understanding of a players' physiological response to training/game demands, future research should incorporate more detailed blood analysis (Lee et al., 2017).

The present study observed moderate associations between CMJ AP, FT, CT and AF to sprint outputs and medium acceleration/deceleration. The CMJ output measure FT demonstrated the greatest sensitivity to all physical match performance parameters with the exception of HAcc ($r = -0.321$ to -0.496, $P = 0.090$ to 0.010). The findings also displayed trends for reduction in AP to TD ($r = 0.313$, $P = 0.099$) and AF to HIN, TSN and MAcc ($r = -0.328$ to -0.349, $P = 0.082$ to 0.063). The magnitude of associations between all other variables was insignificant, trivial or small. These associations therefore support our findings that both concentric outputs and movement strategy can achieve the sensitivity necessary against match external load and the detection of NF in elite footballers.

Lastly, the positional changes 24-h pre to 48-h post-match showed that central midfielders displayed the greatest ES change in Uosm, CK, CMJ height and CMJ FT. In agreement with previous research, this position alongside wide midfielders covered the greatest total distance, HIR distance and HI actions (Bradley et al., 2009). The higher physical output in these positions may explain the greater physiological stress observed 48-h post-match. Central midfielders also covered the greatest number of medium

decelerations that may have contributed to the greater changes in CK concentrations observed due to the high eccentric component in this action know to induce muscle damage (Nosaka and Newton, 2002; Young et al., 2012; de Hoyo et al., 2016). Central defenders displayed the greatest percentage change for decrements in CMJ PP and MF outputs. In this investigation, central defenders covered the lowest distances and high intensity actions. Physical output is more complex than observing distances covered alone, and actions such as kicking, jumping, and heading are powerful movements that are repeatedly performed by players during competitive football, yet are rarely considered as contributors to post-match fatigue. This may also provide reason for the lack of statistical significance and low association between CK and match external load in this investigation. Future research should incorporate a more integrated approach looking at associations between physical, technical and tactical factors that may be associated with changes in recovery-fatigue responses (Bradley and Ade, 2018).

5.5: Conclusion

CK concentrations at 48-h post-match do not seem to be linked to the physical match performance variables. However, CK does seem to be sensitive to the overall demands of match-play that may be associated with 90-min of competitive football. The results of this study also demonstrate that variables related to both performance (FT) and movement strategy (FT:CT, AP) are sensitive to changes in NF 48-h post competitive football in this sub-group of elite senior level football players. The analysis and review of this information post-match may help practitioners determine the readiness of individual players, and aid to design and adjust individual player's recovery strategies.

Chapter 6: Observation of CMJ, CK and Uosm in elite players over the course of a professional football season.

6.1: Introduction

Fatigue is a complex phenomenon with multi-faceted contributors that can present in a multitude of ways from psychological in the brain (e.g. the sensation or perception of tiredness) to physiological in the muscle (e.g. failure in the muscle excitation-contraction coupling process; Abiss and Laursen, 2007; Halson, 2014). In elite football, players suffer from transient, residual, and cumulative fatigue following training and competition (Mohr et al., 2003; Knicker et al., 2011). The observation of recovery-fatigue markers over a relatively short period, such as post-competition or one training week, are unrepresentative of long-term changes. Fatigue can manifest over time, perhaps when the body has not had time to adapt to exercise stress (Selye, 1950; Kentta and Hassmen, 1998). Due to the high frequency and volume of training/competition, recovery time during intensive competition periods may not be long enough to restore physiological homeostasis and may lead to chronic fatigue.

Coaches and practitioners often fear that periods of high exposure may have negative sequelae. Previous studies in elite sport have established that an increase in workload can lead to higher injury incidence (Gabbett, 2004; Gabbett and Jenkins, 2011; Gabbett and Ullah, 2012). Understanding the link between training/match load an injury incidence suggests a valid marker of fatigue should be sensitive to both acute and chronic fluctuations in training and match load (Meeusen et al., 2013). The previous two chapters (4 and 5) have identified that CK concentration, CMJ height, FT, FT:CT, AP and TV are acutely affected 48-h following 90-min of competitive match-play in elite senior football players. However, there is limited research and understanding on the season-long responses in these variables within an elite sporting environment. Longitudinal analyses may aid the understanding of the physiological demands and stress-related alterations induced during a professional football season. This understanding may help practitioners

identify potential signs of chronic fatigue/over-training, and if indicated, adjust individual player's recovery strategies to maintain peak performance throughout the competitive season.

To the author's knowledge, no research to date has examined the long-term fluctuations of CK, CMJ and hydration status over a full competitive season in elite, senior level, football players. Therefore, the aim of this study was to examine the longitudinal fluctuations in physiological and physical performance markers across a season in the English Football League Championship.

6.2: Methods

Participants

Sixteen first-team professional male football players were recruited for this study, which took place during the 2015-16 season as had been previously described in detail in Chapter 3 (section 3.1). Inclusion criteria only included the players who had achieved at least a total of 20 or more competitive league appearances over the course of the season to observe the physiological and physical effects of those who were playing regularly and available for selection (Table 6.1).

Table 6.1. Summary of the recruited players appearances and availability over the season.

	Player Appearances	Total Number of Minutes Played	Games Available	Number of Consecutive Games Started
Player 1	41	3765	41	36
Player 2	38	3340	46	13
Player 3	38	3336	39	16
Player 4	37	3458	42	15
Player 5	36	2699	40	8
Player 6	34	2912	47	15
Player 7	33	2286	44	12
Player 8	30	2717	49	22
Player 9	29	1156	40	4
Player 10	28	2235	29	7
Player 11	28	2517	28	18
Player 12	26	2304	33	11
Player 13	25	1529	30	9
Player 14	24	519	48	1
Player 15	23	1570	47	8
Player 16	22	2014	43	13

Experimental Design

All data were collected as described in Chapter 3 (section 3.2). For this Chapter the data were averaged per week for the recovery-fatigue variables. Due to time constraints, injury or illness, there was a varied number of data sets across the study period and a mean (± SD) of 10.7 ± 2.4 participants per data collection point. Baseline data were collected in a rested state approximately 36-h before the first match of the season (BAS) and on 38 occasions throughout the 40-week season.

Hydration Testing – Urine Osmolality

Hydration testing was measured as described in Chapter 3 (section 3.3.1).

Creatine Kinase Testing

Creatine Kinase testing was measured as described in Chapter 3 (section 3.3.2).

Physical Performance Testing – Counter-movement Jump

The CMJ (a vertical jump test) was performed using a portable force platform (HUR Labs Force Platform 3.8.0.2, Kokkola, Finland) as described in Chapter 3 (section 3.3.3).

Statistical Analysis

The data were analysed by means of the Statistical Package for Social Sciences (SPSS) for Windows (SPSS, Chicago, IL), IBM version 26. All data were log transformed to improve the normality of distribution. After log transformation, the outliers more than three interquartile ranges below the first quartile or above the third quartile were removed from the analysis. To overcome the varied number of data sets across the study period a linear mixed-models procedure was used. Following convention, the alpha level of significance was set at 5 %, hence values where $P < 0.05$ have been referred to as

"significant" and those where $P < 0.10$ but > 0.05 as a "trend" or "marginally significant". When the SPSS output demonstrated significance levels of $P = 0.000$, these were corrected to $P < 0.005$ (Kinear and Gray, 1995). Geometric means were calculated by taking the exponential. Descriptive statistics for the season were calculated from the raw data including the median, interquartile range, 95 % error of margin and % CV from each individual data point prior to log-transformation and the grouping of data per week. After log-transformation a 95 % reference range was derived from the data based on the normal distribution.

6.3: Results

Descriptive statistics from the entire season, with the exception of % CV calculated from group baseline testing data, are presented in Table 6.2. The fluctuation from baseline testing and weeks 1 to 38 over the season are reported in Table 6.3 (6.3.a to 6.3.i) showing the geometric mean, the percentage (%) change to baseline (BAS), statistical significance (P-value) and 95 % confidence intervals (CI). Figures 6.1.a to 6.1.k show the distribution of the data across the season for CMJ, CK and Uosm. Boxplots were used to show the behaviour of these markers throughout the season. Figures 6.2.a to 6.2.k display the position specific changes each week across the season.

Table 6.2. Descriptive statistics, medians (interquartile ranges) and margin of error (95%) for CMJ, CK and Uosm from the entire season. Group % CV from baseline testing.

Measure	Median (IQR)	95 % Reference Interval	Margin of Error (95%)	BAS % CV
Uosm (mOsmols)	300 (240 to 395)	137 to 675	16	51
CK (u/l)	366 (244 to 593)	98 to 1417	45	16
CMJ H (cm)	35 (32 to 38)	28 to 43	0.43	8.7
CT (ms)	837 (774 to 891)	646 to 1075	11.6	9.4
FT (ms)	549 (526 to 575)	491 to 615	3.5	6.0
FT:CT	0.66 (0.61 to 0.72)	0.50 to 0.88	0.01	9.2
MF (N)	1859 (1751 to 1977	1576 to 2210	18.4	11.4
PP (W)	3988 (3791 to 4320)	3342 to 4873	47.9	13.2
AF (N)	1048 (988 to 1087)	839 to 1232	9.8	10.8
AP (W)	1061 (939 to 1197)	768 to 1462	19.4	15.0
TV (m/s)	2.61 (2.51 to 2.72)	2.34 to 2.92	0.02	4.7

Table 6.3.a. Geometric mean (% change to BAS), and P-value (95 % CI) of CMJ, CK and Uosm weekly changes to baseline over a competitive football season.

WEEK	**CK** (u/l)	**HYDR** (mOsmols)	**CMJ H** (cm)	**CMJ CT** (ms)	**CMJ FT** (ms)	**CMJ FT:CT**	**CMJ MF** (N)	**CMJ PP** (W)	**CMJ AF** (N)	**CMJ AP** (W)	**CMJ TV** (m/s)
BAS	280	255	36.8	836.8	557.9	0.67	1841.0	4185.6	1076.8	1125.8	2.68
W1 (AUG)	236 (1.6 %) P = 0.948 (95 % CI: -0.46 to 0.49)	287 (12.6%) P = 0.370 (95 % CI: -0.14 to 0.38)	36.1 (0.5 %) P = 0.855 (95 % CI: -0.05 to 0.06)	811.1 (0.6 %) P = 0.844 (95 % CI: -0.06 to 0.07)	554.6 (0.3 %) P = 0.845 (95 % CI: -0.03 to 0.03)	0.69 (-0.02 %) P = 0.995 (95 % CI: -0.07 to 0.07)	1869.0 (1.6 %) P = 0.491 (95 % CI: -0.03 to 0.06)	4108.2 (1.1 %) P = 0.569 (95 % CI: -0.03 to 0.05)	1081.0 (2.1 %) P = 0.139 (95 % CI: -0.01 to 0.05)	1131.4 (2.9 %) P = 0.569 (95 % CI: -0.07 to 0.13)	2.66 (0.6 %) P = 0.690 (95 % CI: -0.02 to 0.04)
W2 (AUG)	337 (29.4 %) P = 0.237 (95 % CI: -0.17 to 0.69)	270 (4.0 %) P = 0.764 (95 % CI: -0.22 to 0.29)	34.3 (-3.9 %) P = 0.185 (95 % CI: -0.10 to 0.02)	841.7 (3.8 %) P = 0.283 (95 % CI: -0.03 to 0.10)	546.3 (-0.8 %) P = 0.612 (95 % CI: -0.04 to 0.02)	0.65 (-4.7 %) P = 0.246 (95 % CI: -0.13 to 0.03)	1803.1 (0.7 %) P = 0.784 (95 % CI: -0.04 to 0.06)	3944.1 (-0.5 %) P = 0.820 (95 % CI: -0.04 to 0.04)	1052.4 (0.5 %) P = 0.756 (95 % CI: -0.03 to 0.03)	1008.0 (-8.0 %) P = 0.126 (95 % CI: -0.19 to 0.02)	2.58 (-1.8 %) P = 0.289 (95 % CI: -0.05 to 0.02)
W3 (AUG)	270 (-3.1 %) P = 0.889 (95 % CI: -0.47 to 0.41)	301 (11.5 %) P = 0.413 (95 % CI: -0.15 to 0.37)	34.5 (-4.8 %) *P = 0.095* (95 % CI: -0.11 to 0.01)	814.1 (3.3 %) P = 0.329 (95 % CI: -0.03 to 0.10)	542.6 (-1.7 %) P = 0.278 (95 % CI: -0.05 to 0.01)	0.67 (-5.2 %) P = 0.186 (95 % CI: -0.13 to 0.03)	1870.5 (2.0 %) P = 0.426 (95 % CI: -0.03 to 0.07)	4013.0 (-0.6 %) P = 0.743 (95 % CI: -0.05 to 0.03)	1040.4 (-0.3 %) P = 0.862 (95 % CI: -0.03 to 0.03)	1041.7 (-6.7 %) P = 0.178 (95 % CI: -0.17 to 0.03)	2.62 (-1.7 %) P = 0.276 (95 % CI: -0.05 to 0.01)
W4 (AUG)	-	354 (30.0 %) **P = 0.049** (95 % CI: 0.001 to 0.52)	35.6 (-3.7 %) P = 0.190 (95 % CI: -0.09 to 0.02)	837.0 (6.1 %) *P = 0.067* (95 % CI: -0.004 to 0.12)	543.7 (-1.9 %) P = 0.207 (95 % CI: -0.05 to 0.01)	0.65 (-8.0 %) **P = 0.034** (95 % CI: -0.16 to -0.006)	1815.7 (-0.5 %) P = 0.842 (95 % CI: -0.05 to 0.04)	3934.3 (-2.0 %) P = 0.286 (95 % CI: -0.06 to 0.02)	1014.1 (-2.5 %) *P = 0.086* (95 % CI: -0.05 to 0.004)	1000.3 (-9.6 %) *P = 0.050* (95 % CI: -0.20 to -0.0001)	2.62 (-2.2 %) P = 0.169 (95 % CI: -0.05 -to 0.01)

Table 6.3.b. Geometric mean (% change to BAS), and P-value (95 % CI) of CMJ, CK and Uosm weekly changes to baseline over a competitive football season.

WEEK	CK (u/l)	HYDR (mOsmols)	CMJ H (cm)	CMJ CT (ms)	CMJ FT (ms)	CMJ FT:CT	CMJ MF (N)	CMJ PP (W)	CMJ AF (N)	CMJ AP (W)	CMJ TV (m/s)
BASE	280	255	36.8	836.8	557.9	0.67	1841.0	4185.6	1076.8	1125.8	2.68
W5 (SEP)	-	261 (1.7 %) P = 0.909 (95 % CI: -0.27 to 0.30)	35.1 (-4.4 %) P = 0.116 (95 % CI: -0.10 to 0.01)	822.5 (2.4 %) P = 0.446 (95 % CI: -0.04 to 0.09)	545.1 (-2.4 %) P = 0.100 (95 % CI: -0.05 to 0.005)	0.66 (-4.8 %) P = 0.197 (95 % CI: -0.12 to 0.03)	1842.3 (2.2 %) P = 0.348 (95 % CI: -0.02 to 0.07)	3986.5 (-0.9 %) P = 0.634 (95 % CI: -0.05 to 0.03)	1017.9 (-1.8 %) P = 0.206 (95 % CI: -0.05 to 0.01)	1005.2 (-9.4 %) **P = 0.048** (95 % CI: -0.20 to -0.001)	2.63 (-2.4 %) P = 0.129 (95 % CI: -0.05 to 0.01)
W6 (SEP)	339 (16.9 %) P = 0.514 (95 % CI: -0.31 to 0.63)	309 (16.2 %) P = 0.250 (95 % CI: -0.11 to 0.41)	35.9 (-3.7 %) P = 0.202 (95 % CI: -0.09 to 0.02)	828.1 (1.2 %) P = 0.703 (95 % CI: -0.05 to 0.08)	564.1 (0.3 %) P = 0.852 (95 % CI: -0.03 to 0.03)	0.67 (-3.1 %) P = 0.405 (95 % CI: -0.11 to 0.04)	1819.5 (2.2 %) P = 0.347 (95 % CI: -0.02 to 0.07)	4019.6 (0.0 %) P = 0.999 (95 % CI: -0.04 to 0.04)	1014.8 (-0.5 %) P = 0.727 (95 % CI: -0.03 to 0.02)	1034.5 (-4.7 %) P = 0.348 (95 % CI: -0.15 to 0.05)	2.66 (-1.6 %) P = 0.306 (95 % CI: -0.05 to 0.02)
W7 (SEP)	551 (75.5 %) **P = 0.013** (95 % CI: 0.12 to 1.00)	293 (4.7 %) P = 0.757 (95 % CI: -0.25 to 0.34)	34.9 (-6.1 %) **P = 0.026** (95 % CI: -0.12 to -0.007)	814.7 (-0.1 %) P = 0.975 (95 % CI: -0.06 to 0.06)	544.3 (-3.2 %) **P = 0.030** (95 % CI: -0.06 to -0.003)	0.67 (-3.1 %) P = 0.405 (95 % CI: -0.11 to 0.04)	1814.6 (0.2 %) P = 0.920 (95 % CI: -0.04 to 0.05)	4022.7 (-1.6 %) P = 0.379 (95 % CI: -0.05 to 0.02)	1027.3 (-1.1 %) P = 0.419 (95 % CI: -0.04 to 0.02)	1027.2 (-7.3 %) P = 0.128 (95 % CI: -0.17 to 0.02)	2.62 (-3.2 %) **P = 0.035** (95 % CI: -0.06 to -0.002)
W8 (SEP)	427 (45.6 %) P = 0.102 (95 % CI: -0.07 to 0.83)	281 (4.0 %) P = 0.785 (95 % CI: -0.24 to 0.32)	33.5 (-9.2 %) **P = 0.001** (95 % CI: -0.16 to -0.04)	797.5 (-1.9 %) P = 0.573 (95 % CI: -0.08 to 0.05)	544.5 (-2.6 %) P = 0.091 (95 % CI: -0.06 to 0.004)	0.69 (-0.4 %) P = 0.924 (95 % CI: -0.08 to 0.07)	1829.3 (1.9 %) P = 0.439 (95 % CI: -0.03 to 0.07)	3941.6 (-2.6 %) P = 0.179 (95 % CI: -0.07 to 0.01)	1024.1 (-0.6 %) P = 0.711 (95 % CI: -0.03 to 0.02)	1008.5 (-6.5 %) P = 0.201 (95 % CI: -0.17 to 0.04)	2.57 (-4.7 %) **P = 0.003** (95 % CI: -0.08 to -0.02)

Table 6.3.c. Geometric mean (% change to BAS), and P-value (95 % CI) of CMJ, CK and Uosm weekly changes to baseline over a competitive football season.

WEEK	CK (u/l)	HYDR (mOsmols)	CMJ H (cm)	CMJ CT (ms)	CMJ FT (ms)	CMJ FT:CT	CMJ MF (N)	CMJ PP (W)	CMJ AF (N)	CMJ AP (W)	CMJ TV (m/s)
BASE	280	255	36.8	836.8	557.9	0.67	1841.0	4185.6	1076.8	1125.8	2.68
W9 (SEP)	363 (21.2 %) P = 0.429 (95 % CI: -0.29 to 0.67)	308 (16.4 %) P = 0.243 (95 % CI: -0.10 - 0.41)	35.0 (-5.8 %) **P = 0.042** (95 % CI: -0.12 to -0.002)	757.4 (-5.9 %) *P = 0.060* (95 % CI: -0.12 to 0.002)	558.0 (-0.8 %) P = 0.600 (95 % CI: -0.04 to 0.02)	0.74 (5.0 %) P = 0.211 (95 % CI: -0.03 to 0.12)	1923.0 (5.1 %) **P = 0.037** (95 % CI: 0.003 to 0.10)	4156.2 (0.4 %) P = 0.820 (95 % CI: -0.03 to 0.04)	1046.8 (0.5 %) P = 0.744 (95 % CI: -0.02 to 0.03)	1066.8 (-2.1 %) P = 0.672 (95 % CI: -0.12 to 0.08)	2.62 (-3.0 %) *P = 0.058* (95 % CI: -0.06 to 0.001)
W11 (OCT)	320 (4.5 %) P = 0.844 (95 % CI: -0.39 to 0.48)	262 (0.5 %) P = 0.967 (95 % CI: -0.25 to 0.26)	34.8 (-3.9 %) P = 0.159 (95 % CI: -0.10 to 0.02)								
W12 (OCT)	410 (32.0 %) P = 0.226 (95 % CI: -0.17 to 0.73)	291 (11.6 %) P = 0.402 (95 % CI: -0.15 to 0.37)	33.8 (-7.8 %) **P = 0.007** (95 % CI: -0.14 to -0.02)	858.2 (1.1 %) P = 0.734 (95 % CI: -0.05 to 0.08)	537.6 (-2.9 %) *P = 0.059* (95 % CI: -0.06 to 0.001)	0.63 (-4.4 %) P = 0.261 (95 % CI: -0.12 to 0.03)	1879.9 (1.2 %) P = 0.622 (95 % CI: -0.04 to 0.06)	4159.0 (-2.4 %) P = 0.209 (95 % CI: -0.06 to 0.01)	1070.6 (-1.3 %) P = 0.385 (95 % CI: -0.04 to 0.02)	1015.7 (-9.3 %) *P = 0.062* (95 % CI: -0.20 to 0.01)	2.57 (-4.2 %) **P = 0.010** (95 % CI: -0.07 to -0.01)
W13 (OCT)	448 (27.6%) P = 0.316 (95 % CI: -0.23 to 0.72)	276 (10.9 %) P = 0.436 (95 % CI: -0.16 to 0.36)	34.6 (-5.4 %) **P = 0.059** (95 % CI: -0.11 to -0.002)	824.2 (-2.2 %) P = 0.483 (95 % CI: -0.09 to 0.04)	550.9 (-0.7 %) P = 0.622 (95 % CI: -0.04 to 0.02)	0.67 (1.7 %) P = 0.655 (95 % CI: -0.06 to 0.09)	1909.3 (2.9 %) P = 0.216 (95 % CI: -0.02 to 0.08)	4193.7 (-1.3 %) P = 0.489 (95 % CI: -0.05 to 0.02)	1087.4 (0.6 %) P = 0.667 (95 % CI: -0.02 to 0.03)	1100.5 (-1.2 %) P = 0.815 (95 % CI: -0.11 to 0.09)	2.62 (-2.5 %) P = 0.106 (95 % CI: -0.06 to 0.01)

Table 6.3.d. Geometric mean (% change to BAS), and P-value (95 % CI) of CMJ, CK and Uosm weekly changes to baseline over a competitive football season.

WEEK	CK (u/l)	HYDR (mOsmols)	CMJ H (cm)	CMJ CT (ms)	CMJ FT (ms)	CMJ FT:CT	CMJ MF (N)	CMJ PP (W)	CMJ AF (N)	CMJ AP (W)	CMJ TV (m/s)
BASE	280	255	36.8	836.8	557.9	0.67	1841.0	4185.6	1076.8	1125.8	2.68
W14 (NOV)	446 (61.7 %) **P = 0.042** (95 % CI: 0.02 to 0.94)	227 (-8.9 %) P = 0.481 (95 % CI: -0.35 to 0.17)	34.3 (-5.4 %) *P = 0.050* (95 % CI: -0.11 to 0.0001)	829.9 (-0.8 %) P = 0.811 (95 % CI: -0.07 to 0.05)	548.4 (-1.2 %) P = 0.402 (95 % CI: -0.04 to 0.02)	0.66 (-0.2 %) P = 0.958 (95 % CI: -0.08 to 0.07)	1956.0 (6.1 %) **P = 0.010** (95 % CI: 0.01 to 0.10)	4267.5 (1.8 %) P = 0.334 (95 % CI: -0.02 to 0.05)	1091.1 (2.0 %) P = 0.158 (95 % CI: -0.01 to 0.05)	1068.2 (-2.8 %) P = 0.573 (95 % CI: -0.13 to 0.07)	2.62 (-2.0 %) P = 0.196 (95 % CI: -0.05 to 0.01)
W15 (NOV)	478 (117.0 %) **P = 0.004** (95 % CI: 0.24 to 1.31)	246 (-1.4 %) P = 0.921 (95 % CI: -0.29 to 0.26)									
W16 (NOV)	345 (-8.2 %) P = 0.767 (95 % CI: -0.66 to 0.48)	286 (13.1 %) P = 0.353 (95 % CI: -0.14 to 0.38)	35.2 (-1.6 %) P = 0.590 (95 % CI: -0.08 to 0.04)	840.6 (0.1 %) P = 0.974 (95 % CI: -0.06 to 0.07)	560.7 (1.1 %) P = 0.468 (95 % CI: -0.02 to 0.04)	0.67 (0.9 %) P = 0.820 (95 % CI: -0.07 to 0.09)	1996.2 (6.9 %) **P = 0.006** (95 % CI: 0.02 to 0.11)	4344.0 (2.5 %) P = 0.207 (95 % CI: -0.01 to 0.06)	1109.2 (2.0 %) P = 0.190 (95 % CI: -0.01 to 0.05)	1127.7 (1.3 %) P = 0.801 (95 % CI: -0.09 to 0.12)	2.63 (-1.0 %) P = 0.558 (95 % CI: -0.04 to 0.02)
W17 (NOV)	274 (-11.4 %) P = 0.657 (95 % CI: -0.66 to 0.42)	290 (14.2 %) P = 0.299 (95 % CI: -0.12 to 0.38)	35.4 (-3.4 %) P = 0.236 (95 % CI: -0.09 to 0.02)	809.2 (-4.0 %) P = 0.204 (95 % CI: -0.10 to 0.02)	554.3 (-0.1 %) P = 0.939 (95 % CI: -0.03 to 0.03)	0.69 (4.0 %) P = 0.313 (95 % CI: -0.04 to 0.12)	1983.8 (7.0 %) **P = 0.004** (95 % CI: 0.02 to 0.11)	4351.3 (2.4 %) P = 0.213 (95 % CI: -0.01 to 0.06)	1099.8 (2.8 %) *P = 0.071* (95 % CI: -0.002 to 0.06)	1102.1 (0.5 %) P = 0.933 (95 % CI: -0.10 to 0.11)	2.63 (-2.1 %) P = 0.201 (95 % CI: -0.05 to 0.01)

Table 6.3.e. Geometric mean (% change to BAS), and P-value (95 % CI) of CMJ, CK and Uosm weekly changes to baseline over a competitive football season.

WEEK	CK (u/l)	HYDR (mOsmols)	CMJ H (cm)	CMJ CT (ms)	CMJ FT (ms)	CMJ FT:CT	CMJ MF (N)	CMJ PP (W)	CMJ AF (N)	CMJ AP (W)	CMJ TV (m/s)
BASE	280	255	36.8	836.8	557.9	0.67	1841.0	4185.6	1076.8	1125.8	2.68
W18 (DEC)	504 (34.1 %) P = 0.257 (95 % CI: -0.22 to 0.80)	235 (-7.6 %) P = 0.543 (95 % CI: -0.33 to 0.18)	34.1 (-7.2 %) **P = 0.006** (95 % CI: -0.13 to -0.02)	802.2 (-4.0 %) P = 0.175 (95 % CI: -0.10 to 0.02)	548.6 (-2.3 %) P = 0.100 (95 % CI: -0.05 to 0.004)	0.69 (1.9 %) P = 0.595 (95 % CI: -0.05 to 0.09)	1950.2 (6.9 %) **P = 0.003** (95 % CI: 0.02 to 0.11)	4129.8 (-0.5 %) P = 0.799 (95 % CI: -0.04 to 0.03)	1070.7 (1.2 %) P = 0.377 (95 % CI: -0.01 to 0.04)	1078.9 (-3.2 %) P = 0.501 (95 % CI: -0.13 to 0.06)	2.59 (-3.8 %) **P = 0.009** (95 % CI: -0.07 to -0.01)
W19 (DEC)	432 (43.7 %) P = 0.212 (95 % CI: -0.21 to 0.93)	337 (32.6 %) **P = 0.028** (95 % CI: 0.03 to 0.53)	31.6 (-11.3 %) **P = 0.001** (95 % CI: -0.18 to -0.06)	850.5 (3.3 %) P = 0.325 (95 % CI: -0.03 to 0.10)	523.4 (-4.8 %) **P = 0.002** (95 % CI: -0.08 to -0.02)	0.62 (-7.8 %) **P = 0.042** (95 % CI: -0.16 to -0.003)	1842.2 (1.8 %) P = 0.469 (95 % CI: -0.03 to 0.06)	3939.2 (-3.1 %) P = 0.114 (95 % CI: -0.07 to 0.01)	1039.9 (-0.7 %) P = 0.662 (95 % CI: -0.04 to 0.02)	940.7 (-11.5 %) **P = 0.020** (95 % CI: -0.23 to -0.02)	2.49 (-6.0 %) **P = 0.001** (95 % CI: -0.09 to -0.03)
W20 (DEC)		321 (22.3 %) P = 0.139 (95 % CI: -0.07 to 0.47)	34.3 (-6.3 %) **P = 0.027** (95 % CI: -0.12 to -0.01)	851.7 (1.2 %) P = 0.720 (95 % CI: -0.05 to 0.08)	548.9 (-2.2 %) P = 0.152 (95 % CI: -0.05 to 0.008)	0.65 (-3.1 %) P = 0.426 (95 % CI: -0.11 to 0.05)	1827.1 (4.0 %) P = 0.110 (95 % CI: -0.01 to 0.09)	3944.1 (-0.4 %) P = 0.825 (95 % CI: -0.04 to 0.03)	1028.5 (1.0 %) P = 0.488 (95 % CI: -0.02 to 0.04)	1016.8 (-4.7 %) P = 0.364 (95 % CI: -0.15 to 0.06)	2.59 (-3.2 %) **P = 0.046** (95 % CI: -0.07 to -0.001)
W21 (DEC)	670 (158.7 %) **P = 0.001** (95 % CI: 0.48 to 1.42)	353 (33.2 %) **P = 0.047** (95 % CI: 0.004 to 0.57)									

Table 6.3.f. Geometric mean (% change to BAS), and P-value (95 % CI) of CMJ, CK and Uosm weekly changes to baseline over a competitive football season.

WEEK	CK (u/l)	HYDR (mOsmols)	CMJ H (cm)	CMJ CT (ms)	CMJ FT (ms)	CMJ FT:CT	CMJ MF (N)	CMJ PP (W)	CMJ AF (N)	CMJ AP (W)	CMJ TV (m/s)
BASE	280	255	36.8	836.8	557.9	0.67	1841.0	4185.6	1076.8	1125.8	2.68
W23 (JAN)	538 (104.6 %) **P = 0.003** (95 % CI: 0.25 to 1.18)	295 (13.5 %) P = 0.341 (95 % CI: -0.13 to 0.39)	33.6 (-8.8 %) **P = 0.001** (95 % CI: -0.15 to -0.04)	841.7 (4.6 %) P = 0.146 (95 % CI: -0.02 to 0.11)	539.7 (-4.0 %) **P = 0.005** (95 % CI: -0.07 to -0.01)	0.64 (-8.4 %) **P = 0.019** (95 % CI: -0.16 to -0.01)	1899.6 (5.3 %) **P = 0.022** (95 % CI: 0.01 to 0.10)	4039.7 (0.4 %) P = 0.838 (95 % CI: -0.03 to 0.04)	1044.2 (0.9 %) P = 0.502 (95 % CI: -0.02 to 0.04)	1024.1 (-6.8 %) P = 0.149 (95 % CI: -0.17 to 0.03)	2.57 (-4.6 %) **P = 0.002** (95 % CI: -0.08 to -0.02)
W24 (JAN)	380 (25.7 %) P = 0.295 (95 % CI: -0.20 to 0.66)	325 (23.7 %) P = 0.111 (95 % CI: -0.05 to 0.47)	35.2 (-4.5 %) P = 0.108 (95 % CI: -0.10 to 0.01)	850.6 (4.6 %) P = 0.157 (95 % CI: -0.02 to 0.11)	550.7 (-2.2 %) P = 0.128 (95 % CI: -0.05 to 0.006)	0.65 (-6.5 %) *P = 0.077* (95 % CI: -0.14 to 0.007)	1883.0 (2.7 %) P = 0.246 (95 % CI: -0.02 to 0.07)	4129.6 (0.6 %) P = 0.765 (95 % CI: -0.03 to 0.04)	1063.9 (1.0 %) P = 0.475 (95 % CI: -0.02 to 0.04)	1077.9 (-3.9 %) P = 0.426 (95 % CI: -0.14 to 0.06)	2.62 (-2.6 %) *P = 0.094* (95 % CI: -0.06 to 0.004)
W25 (JAN)	358 (32.5 %) P = 0.234 (95 % CI: -0.18 to 0.75)	498 (84.4 %) **P = 0.001** (95 % CI: 0.29 to 0.93)	35.9 (-3.6 %) P = 0.214 (95 % CI: -0.09 to 0.02)	839.8 (5.2 %) P = 0.131 (95 % CI: -0.02 to 0.12)	558.9 (-1.2 %) P = 0.444 (95 % CI: -0.04 to 0.02)	0.67 (-5.4 %) P = 0.162 (95 % CI: -0.13 to 0.02)	1865.4 (3.0 %) P = 0.206 (95 % CI: -0.02 to 0.08)	4054.7 (0.6 %) P = 0.752 (95 % CI: -0.03 to 0.04)	1038.3 (0.6 %) P = 0.684 (95 % CI: -0.02 to 0.04)	1149.2 (3.4 %) P = 0.515 (95 % CI: -0.07 to 0.13)	2.65 (-1.9 %) P = 0.220 (95 % CI: -0.05 to 0.01)
W26 (JAN)	269 (-3.6 %) P = 0.886 (95 % CI: -0.54 to 0.47)	596 (131.2 %) **P = 0.001** (95 % CI: 0.58 to 1.10)	36.8 (-1.4 %) P = 0.634 (95 % CI: -0.07 to 0.04)	811.3 (3.2 %) P = 0.353 (95 % CI: -0.03 to 0.10)	564.5 (-0.3 %) P = 0.858 (95 % CI: -0.03 to 0.03)	0.70 (-3.4 %) P = 0.394 (95 % CI: -0.11 to 0.04)	1810.9 (0.9 %) P = 0.722 (95 % CI: -0.04 to 0.06)	4052.5 (1.5 %) P = 0.439 (95 % CI: -0.02 to 0.05)	1035.7 (1.8 %) P = 0.222 (95 % CI: -0.01 to 0.05)	1102.5 (0.8 %) P = 0.885 (95 % CI: -0.10 to 0.11)	2.71 (0.1 %) P = 0.948 (95 % CI: -0.03 to 0.03)

Table 6.3.g. Geometric mean (% change to BAS), and P-value (95 % CI) of CMJ, CK and Uosm weekly changes to baseline over a competitive football season.

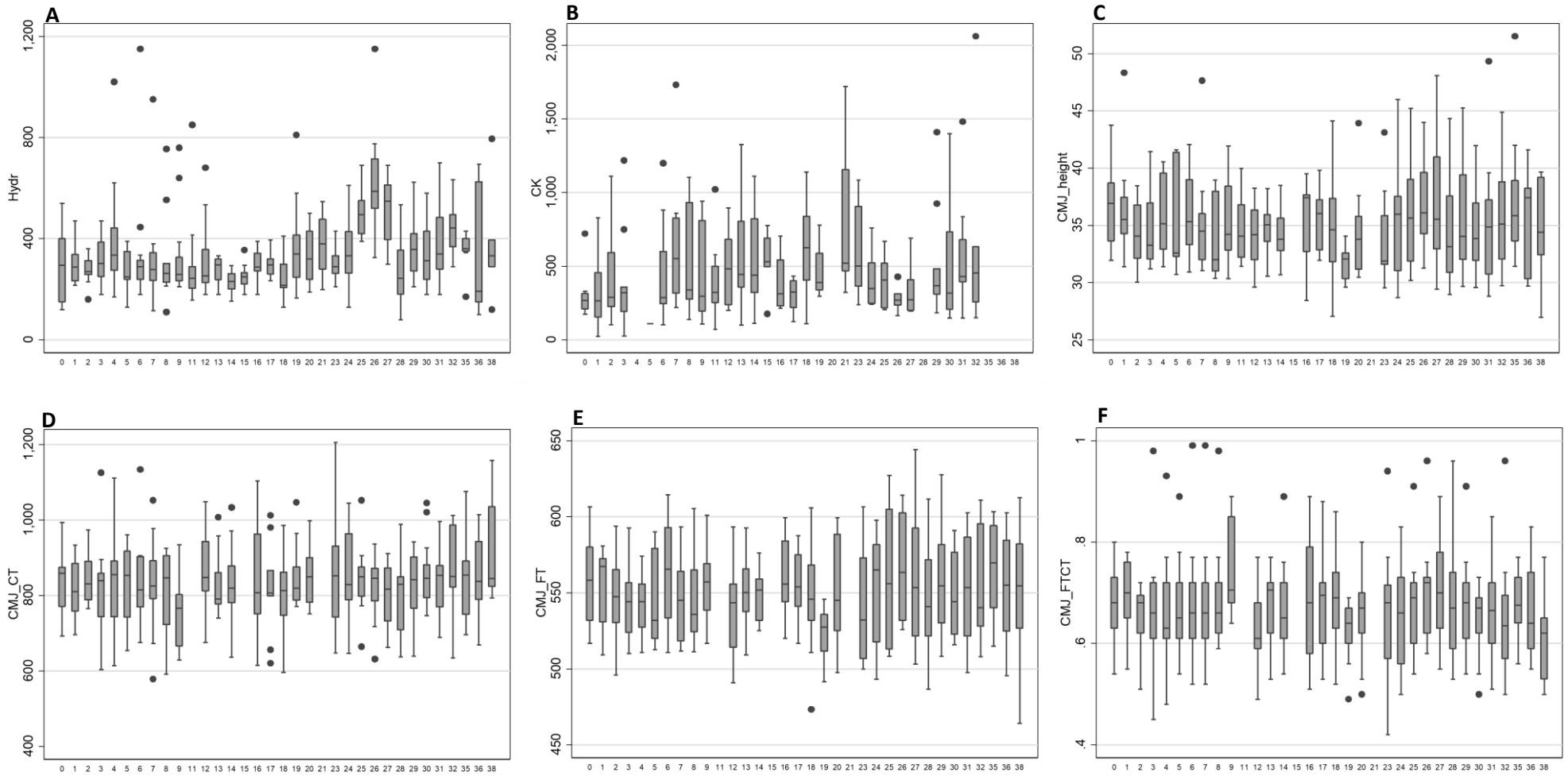
WEEK	CK (u/l)	HYDR (mOsmols)	CMJ H (cm)	CMJ CT (ms)	CMJ FT (ms)	CMJ FT:CT	CMJ MF (N)	CMJ PP (W)	CMJ AF (N)	CMJ AP (W)	CMJ TV (m/s)
BASE	280	255	36.8	836.8	557.9	0.67	1841.0	4185.6	1076.8	1125.8	2.68
W27 (FEB)	298 (19.1 %) P = 0.435 (95 % CI: -0.27 to 0.62)	492 (80.3 %) **P = 0.001** (95 % CI: 0.30 to 0.88)	36.2 (-3.2 %) P = 0.253 (95 % CI: -0.09 to 0.02)	799.5 (3.0 %) P = 0.348 (95 % CI: -0.03 to 0.09)	556.5 (-1.5 %) P = 0.318 (95 % CI: -0.04 to 0.01)	0.70 (-4.4 %) P = 0.242 (95 % CI: -0.12 to 0.03)	1904.2 (3.8 %) P = 0.111 (95 % CI: -0.01 to 0.08)	4059.3 (0.4 %) P = 0.837 (95 % CI: -0.03 to 0.04)	1055.5 (1.3 %) P = 0.352 (95 % CI: -0.01 to 0.04)	1132.1 (-1.1 %) P = 0.826 (95 % CI: -0.11 to 0.09)	2.67 (-1.8 %) P = 0.256 (95 % CI: -0.05 to 0.01)
W28 (FEB)		229 (-16.0 %) P = 0.202 (95 % CI: -0.44 to 0.09)	34.3 (-7.7 %) **P = 0.004** (95 % CI: -0.13 to -0.03)	801.5 (0.7 %) P = 0.831 (95 % CI: -0.06 to 0.07)	543.8 (-3.6 %) **P = 0.012** (95 % CI: -0.07 to -0.01)	0.68 (-4.4 %) P = 0.241 (95 % CI: -0.12 to 0.03)	1851.2 (1.5 %) P = 0.514 (95 % CI: -0.03 to 0.06)	3964.4 (-2.7 %) P = 0.151 (95 % CI: -0.06 to 0.01)	1047.5 (1.0 %) P = 0.482 (95 % CI: -0.02 to 0.04)	1087.8 (-3.1 %) P = 0.525 (95 % CI: -0.13 to 0.07)	2.60 (-4.2 %) **P = 0.006** (95 % CI: -0.07 to -0.01)
W29 (FEB)	417 (25.7 %) P = 0.322 (95 % CI: -0.23 to 0.68)	356 (30.4 %) **P = 0.039** (95 % CI: 0.01 to 0.52)	35.3 (-4.8 %) *P = 0.066* (95 % CI: -0.10 to 0.003)	830.4 (2.8 %) P = 0.360 (95 % CI: -0.03 to 0.09)	554.4 (-1.8 %) P = 0.204 (95 % CI: -0.04 to 0.01)	0.67 (-4.5 %) P = 0.203 (95 % CI: -0.12 to 0.02)	1888.1 (4.0 %) *P = 0.068* (95 % CI: -0.002 to 0.08)	4062.9 (0.1 %) P = 0.976 (95 % CI: -0.03 to 0.04)	1061.0 (1.9 %) P = 0.158 (95 % CI: -0.01 to 0.05)	1108.4 (-1.0 %) P = 0.832 (95 % CI: -0.10 to 0.08)	2.63 (-2.7 %) *P = 0.068* (95 % CI: -0.06 to 0.002)
W30 (FEB)	362 (23.1 %) P = 0.343 (95 % CI: -0.22 to 0.64)	315 (17.8 %) P = 0.194 (95 % CI: -0.08 to 0.41)	34.2 (-6.7 %) **P = 0.011** (95 % CI: -0.12 to -0.02)	854.1 (4.8 %) P = 0.121 (95 % CI: -0.01 to 0.11)	548.2 (-2.3%) *P = 0.091* (95 % CI: -0.05 to 0.003)	0.64 (-6.4 %) *P = 0.066* (95 % CI: -0.14 to 0.004)	1868.9 (3.0 %) P = 0.178 (95 % CI: -0.01 to 0.07)	4050.7 (-0.03 %) P = 0.987 (95 % CI: -0.03 to 0.03)	1050.4 (0.7 %) P = 0.603 (95 % CI: -0.02 to 0.03)	1037.8 (-6.7 %) P = 0.145 (95 % CI: -0.16 to 0.02)	2.59 (-3.6 %) **P = 0.013** (95 % CI: -0.07 to -0.01)

Table 6.3.h. Geometric mean (% change to BAS), and P-value (95 % CI) of CMJ, CK and Uosm weekly changes to baseline over a competitive football season.

WEEK	CK (u/l)	HYDR (mOsmols)	CMJ H (cm)	CMJ CT (ms)	CMJ FT (ms)	CMJ FT:CT	CMJ MF (N)	CMJ PP (W)	CMJ AF (N)	CMJ AP (W)	CMJ TV (m/s)
BASE	280	255	36.8	836.8	557.9	0.67	1841.0	4185.6	1076.8	1125.8	2.68
W31 (FEB/ MAR)	471 (40.9 %) P = 0.135 (95 % CI: -0.11 to 0.79)	355 (33.4 %) **P = 0.023** (95 % CI: 0.04 to 0.53)	34.6 (-6.6 %) **P = 0.013** (95 % CI: -0.12 to -0.01)	836.4 (3.7 %) P = 0.237 (95 % CI: -0.02 to 0.10)	550.9 (-2.4 %) *P = 0.083* (95 % CI: -0.05 to 0.003)	0.66 (-5.6 %) P = 0.123 (95 % CI: -0.13 to 0.02)	1811.4 (0.6 %) P = 0.782 (95 % CI: -0.04 to 0.05)	3986.5 (-1.0 %) P = 0.565 (95 % CI: -0.05 to 0.03)	1040.2 (0.8 %) P = 0.583 (95 % CI: -0.02 to 0.03)	1063.1 (-4.1 %) P = 0.390 (95 % CI: -0.14 to 0.05)	2.61 (-3.5 %) **P = 0.018** (95 % CI: -0.07 to -0.01)
W32 (MAR)	438 (18.5 %) P = 0.489 (95 % CI: -0.31 to 0.65)	428 (55.7 %) **P = 0.001** (95 % CI: 0.18 to 0.70)	35.5 (-5.8 %) **P = 0.032** (95 % CI: -0.11 to -0.01)	865.8 (5.3 %) *P = 0.096* (95 % CI: -0.01 to 0.11)	554.5 (-2.7 %) *P = 0.056* (95 % CI: -0.06 to 0.0007)	0.64 (-7.5 %) **P = 0.036** (95 % CI: -0.15 to -0.005)	1840.3 (2.2 %) P = 0.339 (95 % CI: -0.02 to 0.07)	4071.2 (0.7 %) P = 0.701 (95 % CI: -0.03 to 0.04)	1034.0 (1.0 %) P = 0.481 (95 % CI: -0.02 to 0.04)	1038.5 (-6.2 %) P = 0.189 (95 % CI: -0.16 to 0.03)	2.64 (-3.0 %) **P = 0.046** (95 % CI: -0.06 to -0.001)
W35 (APR)		349 (33.0 %) **P = 0.047** (95 % CI: 0.003 to 0.57)	36.8 (-2.6 %) P = 0.364 (95 % CI: -0.08 to 0.03)	840.8 (2.2 %) P = 0.497 (95 % CI: -0.04 to 0.09)	563.2 (-1.4 %) P = 0.339 (95 % CI: -0.04 to 0.02)	0.67 (-3.5 %) P = 0.356 (95 % CI: -0.11 to 0.04)	1905.5 (3.8 %) P = 0.118 (95 % CI: -0.009 to 0.08)	4192.4 (0.1 %) P = 0.981 (95 % CI: -0.04 to 0.04)	1051.3 (0.2 %) P = 0.876 (95 % CI: -0.03 to 0.03)	1088.8 (-3.2 %) P = 0.522 (95 % CI: -0.13 to 0.07)	2.69 (-1.5 %) P = 0.345 (95 % CI: -0.05 to 0.02)
W36 (APR)		262 (-2.7 %) P = 0.856 (95 % CI: -0.32 to 0.27)	35.6 (-5.7 %) **P = 0.040** (95 % CI: -0.11 to -0.002)	849.0 (4.5 %) P = 0.169 (95 % CI: -0.02 to 0.11)	554.4 (-2.9 %) **P = 0.045** (95 % CI: -0.06 to -0.0007)	0.65 (-6.8 %) *P = 0.065* (95 % CI: -0.15 to 0.004)	1890.0 (4.2 %) *P = 0.073* (95 % CI: -0.004 to 0.09)	4124.3 (0.7 %) P = 0.701 (95 % CI: -0.03 to 0.04)	1028.7 (0.3 %) P = 0.837 (95 % CI: -0.02 to 0.03)	975.2 (-10.8 %) **P = 0.023** (95 % CI: -0.21 to -0.02)	2.64 (-3.1 %) **P = 0.048** (95 % CI: -0.06 to -0.0003)

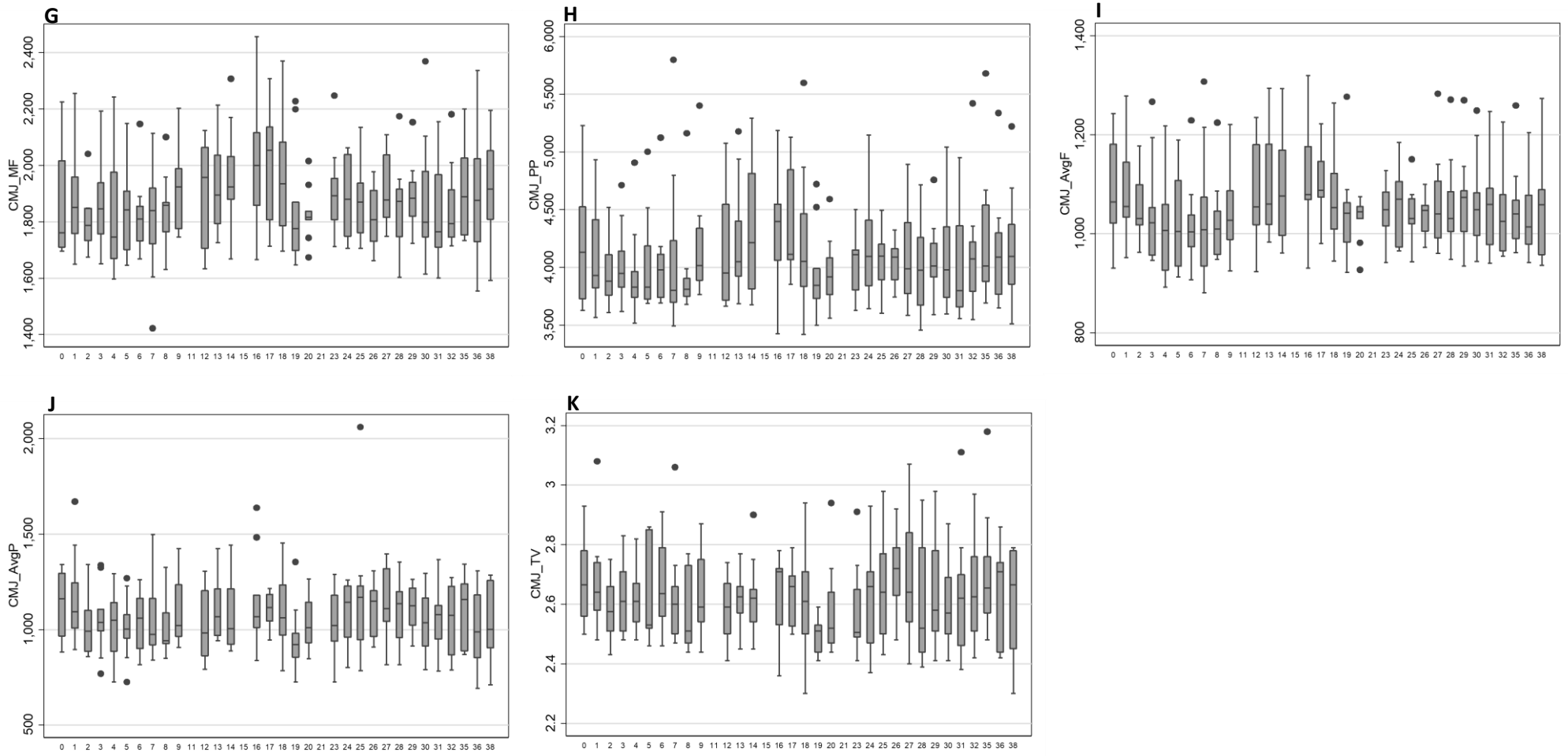
Table 6.3.i: Geometric mean (% change to BAS), and P-value (95 % CI) of CMJ, CK and Uosm weekly changes to baseline over a competitive football season.

WEEK	**CK** (u/l)	**HYDR** (mOsmols)	**CMJ H** (cm)	**CMJ CT** (ms)	**CMJ FT** (ms)	**CMJ FT:CT**	**CMJ MF** (N)	**CMJ PP** (W)	**CMJ AF** (N)	**CMJ AP** (W)	**CMJ TV** (m/s)
BASE	280	255	36.8	836.8	557.9	0.67	1841.0	4185.6	1076.8	1125.8	2.68
W38 (APR)		325 (14.0 %) P = 0.453 (95 % CI: -0.21 to 0.47)	34.9 (-8.8 %) **P = 0.001** (95 % CI: -0.15 to -0.04)	909.1 (10.2 %) **P = 0.003** (95 % CI: 0.03 to 0.16)	549.1 (-4.1 %) **P = 0.006** (95 % CI: -0.07 to -0.01)	0.61 (-12.4 %) **P = 0.001** (95 % CI: -0.21 to -0.06)	1893.4 (2.5 %) P = 0.289 (95 % CI: -0.02 to 0.07)	4148.2 (-2.2 %) P = 0.244 (95 % CI: -0.06 to 0.02)	1054.5 (-0.3 %) P = 0.842 (95 % CI: -0.03 to 0.03)	1013.2 (-10.9 %) **P = 0.025** (95 % CI: -0.22 to -0.01)	2.61 (-5.0 %) **P = 0.002** (95 % CI: -0.08 to -0.02)



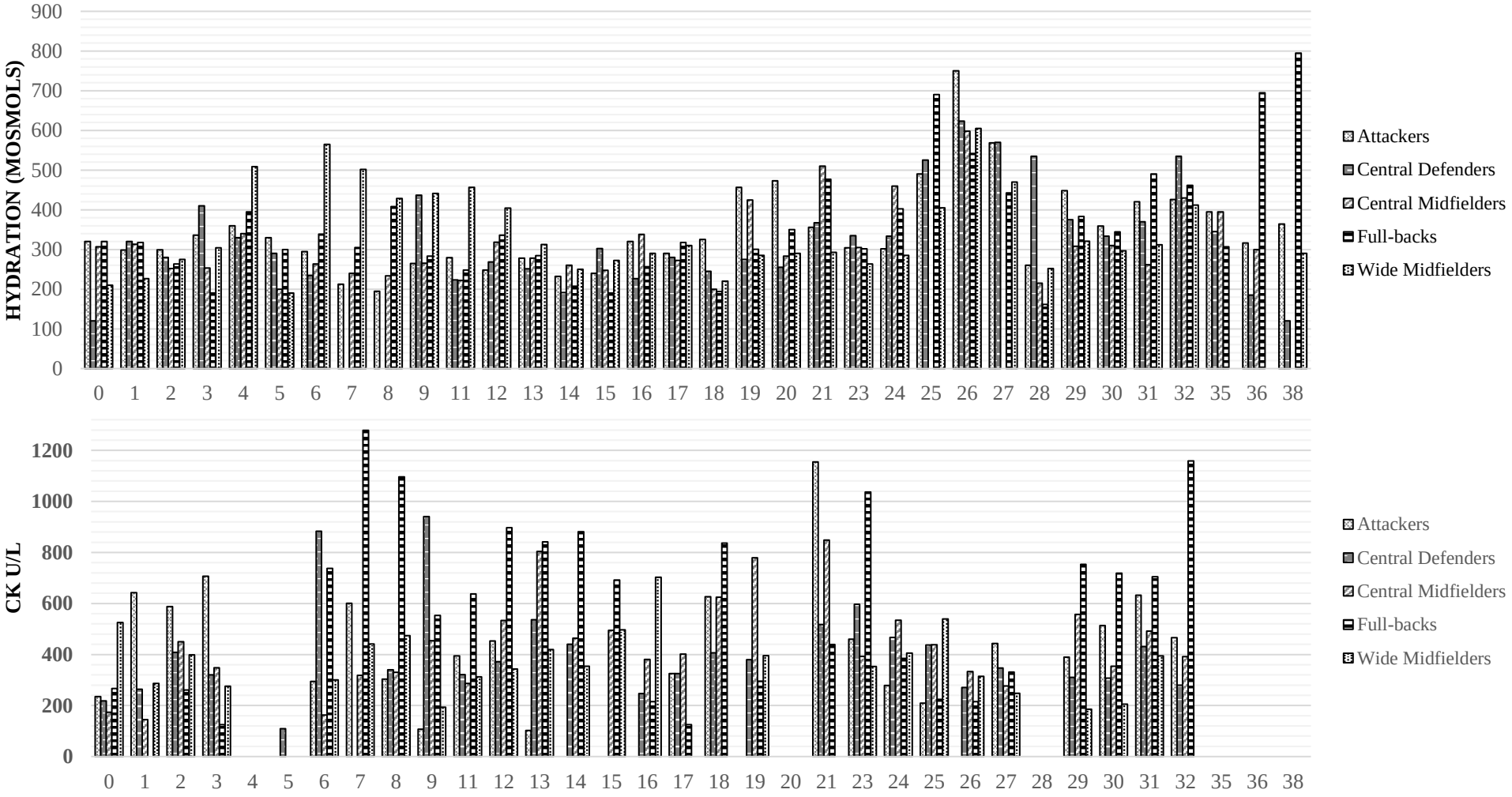
Figures 6.1.a – 6.1.f. Boxplots of recovery-fatigue markers over the course of a competitive season (week 1 -38).

• *Denotes outlier*

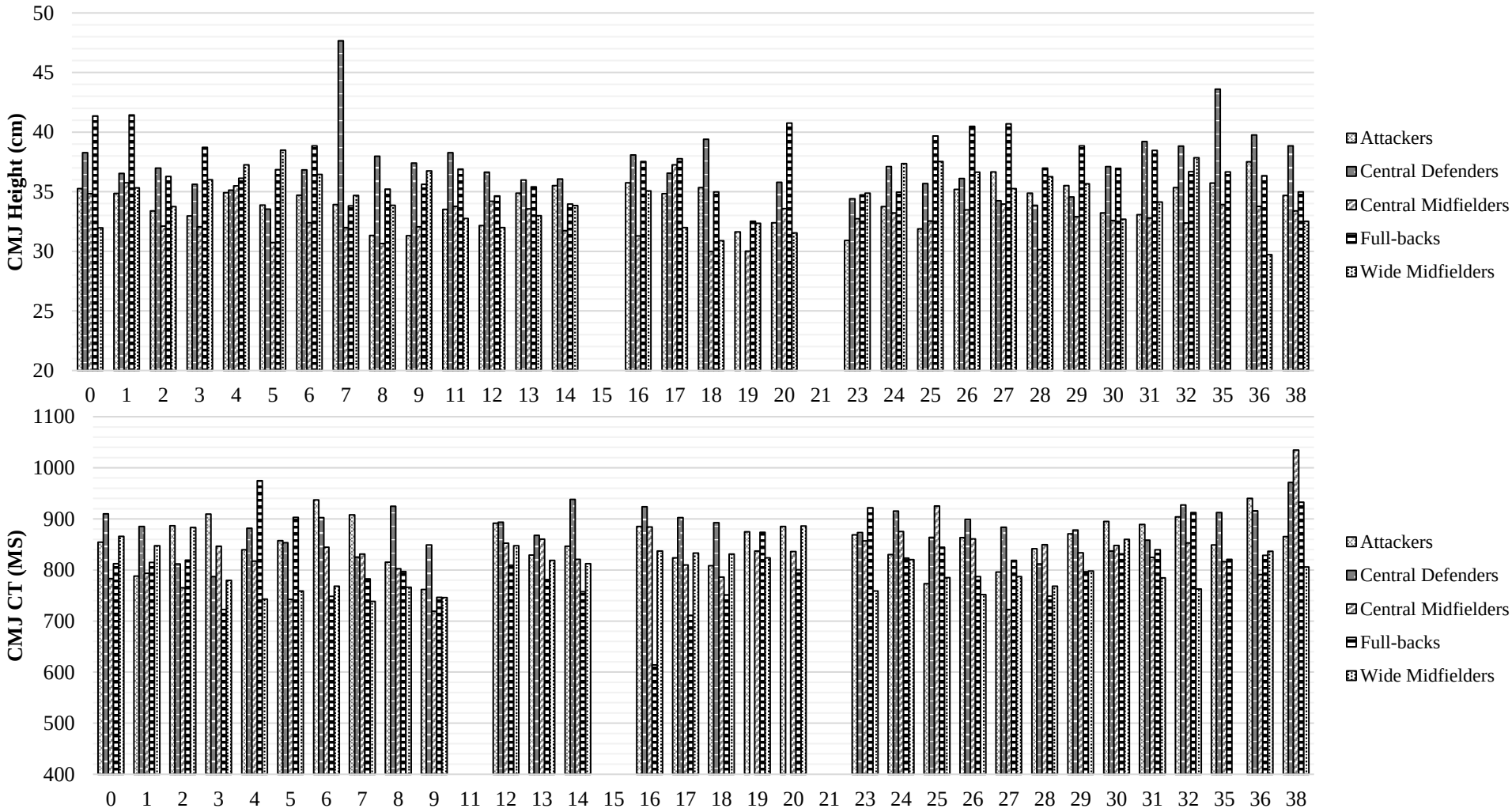


Figures 6.1.g – 6.1.k. Boxplots of recovery-fatigue markers over the course of a competitive season (week 1 -38)

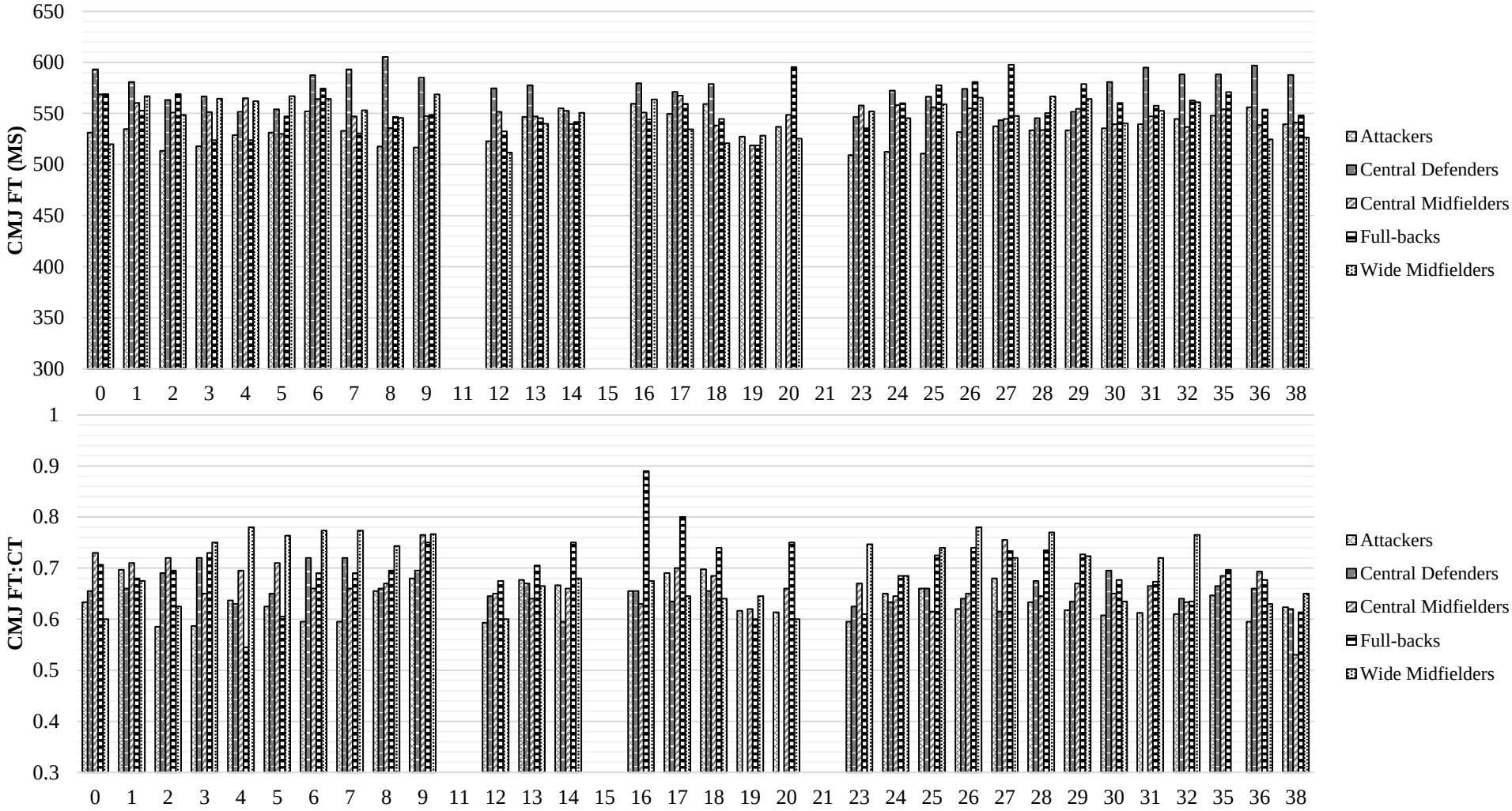
•*Denotes outlier*



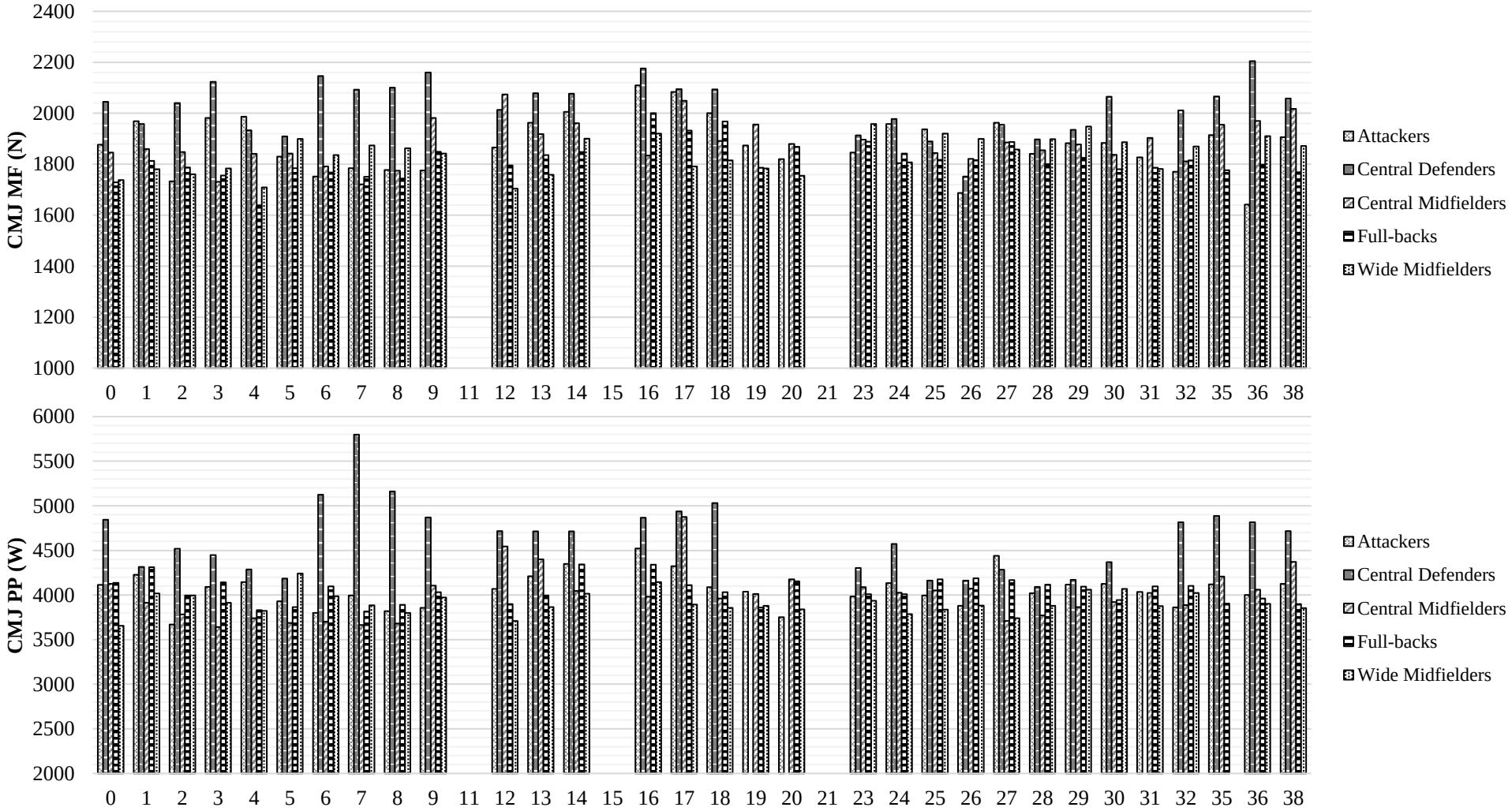
Figures 6.2.a – 6.2.b. Weekly average positional differences across the season for Uosm (mOsmols) and CK concentrations (u/l).



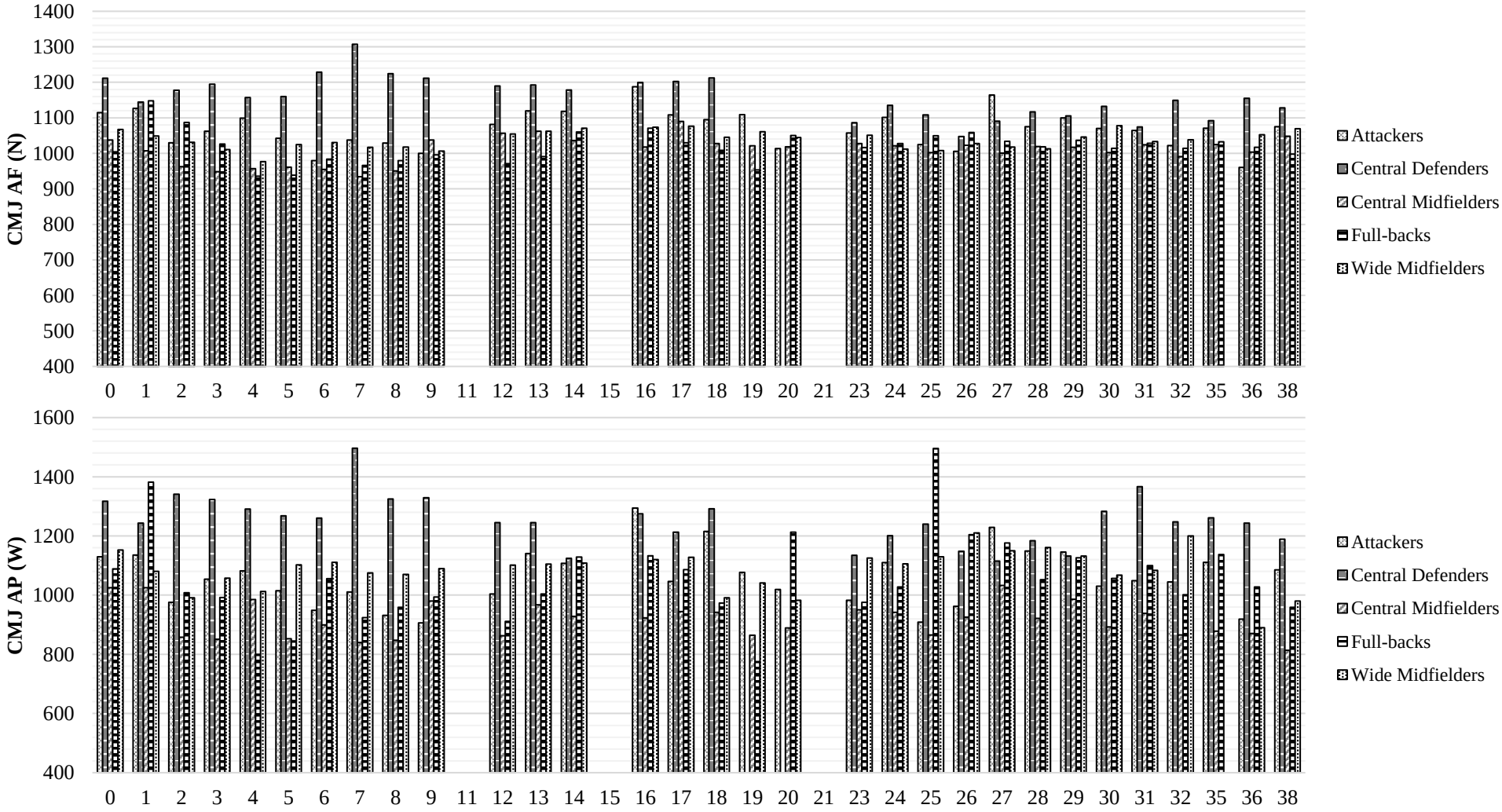
Figures 6.2.c – 6.2.d. Weekly average positional differences across the season for CMJ height (cm) and CT (ms).



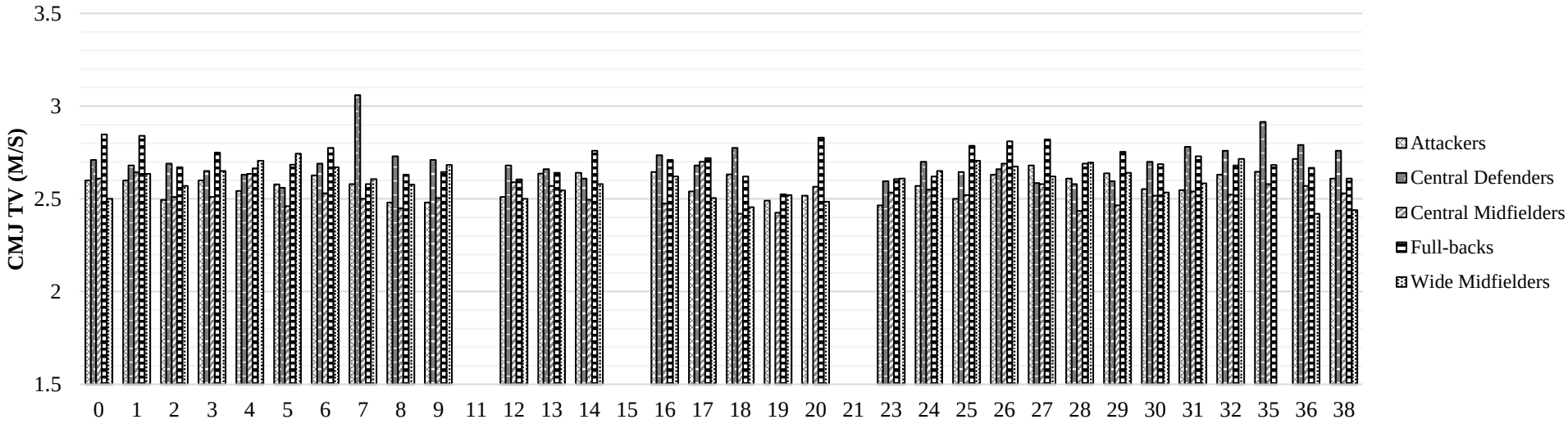
Figures 6.2.e – 6.2.f. Weekly average positional differences across the season for CMJ FT (ms) and FT:CT ratio.



Figures 6.2.g – 6.2.h. Weekly average positional differences across the season for CMJ MF (N) and CMJ PP (W).



Figures 6.2.i – 6.2.j. Weekly average positional differences across the season for CMJ AF (N) and CMJ AP (W).



Figures 6.2.k Weekly average positional differences across the season for CMJ TV (m/s)

Hydration – Urine Osmolality

Uosm levels were statistically significant between weeks (P = 0.001), where 10 out of 33 time-points were significantly higher than baseline over the season (30 %). Week 26 displayed the greatest increase to baseline (131 %, P = 0.001). Statistically significant increases were also observed at weeks 4, 19, 21, 25, 27, 29, 31, 32, and 35 in comparison to baseline measures (30 to 84 %, P < 0.05; Table 6.3).

Creatine Kinase

Creatine Kinase levels were statistically significant between weeks (P = 0.001), where CK concentrations were significantly higher than baseline at 5 out of 26 of time points over the season (19 %). The greatest increase was observed at week 21 (159 %, P = 0.001). Week 7, 14 to 15, and 23 also displayed statistically significant increases compared to baseline (62 to 117 %, P < 0.05; Table 6.3).

Physical Performance – Countermovement Jump

Table 6.3 shows that CMJ height displayed the greatest variation to baseline with 14 out of 31 (45.2 %) of time points demonstrating statistically significant decreases to baseline measures (weeks 7, 8, 9, 12, 18, 19, 20, 23, 28, 30, 31, 32, 36, and 38 [-5.4 to -11.3 %, P < 0.05]). The greatest impairment in CMJ height was observed at week 19 (-11.3 %, P = 0.001). Week 19 also demonstrated statistically significant changes to baseline in CMJ FT, FT:CT, AP and TV (-4.8 to -11.5 %, P < 0.05). Similarly, week 23 displayed reductions in CMJ height, FT, FT:CT, and TV (-4.0 % to -8.8 %, P < 0.05), but increases in CMJ MF (5.3 %, P = 0.022). Week 38 was the only other week show substantial changes to baseline in several CMJ variables (height, FT, CT, FT:CT, AP, TV). Week 38 was the only week CMJ CT demonstrated a statistically significant to baseline (3.3 %, P < 0.001; Table 6.3).

CMJ TV was the next successive CMJ output to display high variations to baseline with 13 out of 30 (43.3%) of time points demonstrating significant decreases to baseline measures (week 7, 8, 12, 18, 19, 20, 23, 28, 30, 31, 32, 36, and 38 [-3.0 to -6.0 %, $P < 0.05$]). The CMJ output measure FT was statistically significantly lower than baseline at 6 out of 30 (20.0 %) of time points over the season (week 7, 19, 23, 28, 36 and 38 [-2.9 to -4.8 %, $P < 0.05$]). Further, CMJ FT:CT was statistically significant from baseline at 5 out of 30 (16.7 %) of time points (weeks 4, 19, 23, 32, and 38 [-7.5 to -12.4 %, $P < 0.05$]) and CMJ AP was statistically significant from baseline at 4 out of 30 (13.3 %) of time points (weeks 5, 19, 36 and 38 [-9.4 to -11.5 %, $P < 0.05$]). Statistically significant increases to baseline were observed in CMJ MF at 6 out of 30 (20.0 %) of time points over the season (weeks 9, 14, 16, 17, 18, 23 [5.1 to 7.0 %, $P < 0.05$]).

Table 6.2 shows there was no statistically significant change to baseline observed in CMJ PP and AF over the course of the season (-3.1 to 2.5 %, -2.5 to 2.8 %, $P > 0.05$, respectively).

6.4: Discussion

The main findings of the study were that CMJ max force can be increased and/or maintained over a season despite alterations in jump strategy that may be indicative of low-lowel NF. Large increases in CK concentrations compared to baseline were also observed, but minimal fluctuation was observed over the season. Further, changes in hydration levels due to training/match-play may not be significant enough to induce detrimental performance changes beyond the sensitivity of the measuring device. To the author's knowledge, this is the first study to investigate weekly variations in these recovery-fatigue measures over a competitive season in elite football players.

Plasma CK concentrations have been widely adopted as an indicator of tissue damage/muscular stress in elite sport, but due to the large inter-individual variability in this biomarker it has been questioned for its use as a valid marker of recovery (Mougios, 2007; Mahmutyazicioglu et al., 2018). As expected our observed reference range (95 %) for CK across the season (98 to 1417 u/l) was greater in comparison to CK from male non-athletes in a rested state (45 to 491 u/l; Mougios, 2007), but similar to previous reported reference ranges in elite football players (65 to 1972 u/l; Mahmutyazicioglu et al., 2018). The median CK concentration from the entire season (366 u/l) is somewhat higher than observations previously reported (178 u/l; Nowakowska et al., 2019). However, this may have been attributable to our sub-group of elite football players used. For example, the expression of CK can be influenced by factors such as ethnicity, age and fibre type composition (Brewster et al., 2007). Due to the small sample size in the current study (n =16) I did not take into account race/ethnicity, however Brewster et al. (2007) confirmed that naturally higher levels of plasma CK are found in the black population in comparison to individuals of either white Caucasian or South Asian descent. Cultural diversity among the footballing population is commonplace, with 31 %

(5 out of 16) of our sub-group of black ethnicities who may have held higher than average CK levels that could have influenced the groups median (Mahmutyazicioglu et al., 2018). The intra-individual variability in CK testing in a rested stated has been reported between 18.5 to 20 % in football players (Harper et al., 2016; Chrismas et al., 2017), which was similar to our group baseline testing of 16 %. The high inter-individual variability in CK concentration should be accounted for when practitioners are utilising CK values to determine the presence of muscle damage to inform training/recovery. Changes in CK concentrations should be compared to individual baseline measures and only comparisons between groups should be used when of similar race/ethnicity.

In the present study, CK concentrations showed a significant increase compared to baseline values at 5 out of 32 time points (19 %), all of which were recorded in the first half of the competitive season and above the % CV discussed. As the season progresses it would be expected to observe reductions in CK concentrations related to the 'repeated bout effect'. Observations by Lazarim et al. (2009) may provide results indicative of a positive muscular adaptive response in football players during a Brazilian national championship. The concentration of CK during the month of August, at the beginning of the competitive season, was significantly higher to that of September, October and November. Moreover, the distribution of the group's median CK activity decreased as the season progressed. It could be suggested that due to the superior athletic status of professional footballers, they have an increased tolerance to load and their muscles are able to cope with the higher demands placed upon them without reaching 'breaking point', resulting in minimal disruption of skeletal muscle membrane in comparison to players of lower-level or non-athletes (Hoffman et al., 2005). This muscular adaptation involves neurological and cellular factors, which increases the protection of tissue against further damage (Clarkson et al., 1992) and may explain the decrease in CK concentration

in the study aforementioned and the minimal fluctuation of plasma CK concentrations observed throughout the season in the present study.

The observation of CK for monitoring physiological response may have a threshold for applicability, such that when players reach a certain level of exposure to training and matches, CK testing lacks sensitivity to identify physiological coping and exercise stress. Nevertheless, previous research cannot easily be compared with the present study because of the irregularity of testing time points. Only one study, comparable to our own, undertook repeated blood analyses, 24-h after each match, over an entire competitive season in elite Polish football players (Nowakowska et al., 2019). A weak-positive correlation between CK concentration and cumulative match-time in midfielders/defenders was observed. The positional differences in match-related activity has previously been reported showing the greatest total distance is covered by central and wide midfielders (Bradley et al., 2009). Therefore, the increased trend in CK levels in this sub-group is to be expected due their increased physical output but it does not support the notion of muscular adaptation in elite football players as previously discussed.

The high variability in CK concentrations within and between football players combined with inconsistent findings on the validity of CK highlights the importance of utilising more than one biomarker when attempting to detect over-training and/or fatigue (Lee et al., 2017). Other blood biomarkers (aspartate aminotransferase, lactate dehydrogenase, and creatinine) have shown to be reliable indicators of recovery due to the lower inter-individual variability in these parameters in comparison to CK (Nowakowska et al., 2019). Researchers have agreed multiple biomarkers should be measured together, thus future research should incorporate more detailed blood analyses on chronic inflammation

and muscle damage to aid interpretation of a players physiological response to training and match-play (Lee et al., 2017).

The results from the current study observed alterations in both CMJ movement strategy and concentric derived outputs. Jump height displayed the greatest variation to observed baseline values with 14 out 31 of time points (45 %) statistically lower. The magnitude of change during these time points ranged from 5.4 to 11.3 %, which is outside the CV % reported in previous research (3.8 to 5 %; Cormack et al., 2008a; Gathercole et al., 2015, Thorpe et al., 2015). Although CMJ height is the most commonly collected and reported variable due to its simplicity, it has been criticised for its use in the detection of NF due to its concentric focused nature. Meister et al. (2013) observed a tendency towards higher CMJ heights during a three-week period of high match exposure in elite German football players. However, the lack of observed relationship could be a consequence of the low threshold used for the high match exposure group; > 270-min could arguably be too low to induce signs of overload. Furthermore, Thorpe et al. (2017) observed a non-significant trivial to small relationship between changes in CMJ height and THIR distance accumulated over two to four days, indicating that the sensitivity of CMJ height to changes in training load is not improved when compared with training loads beyond the previous day of training. Previous research has suggested that alternative CMJ measures may provide more sensitivity to alterations in load than CMJ height (Balloch, 2018).

The output variable FT:CT has been suggested as an effective marker for detecting NF due to its reflection of outputs concerning concentric push-off (FT) and altered movement strategy (CT). In the present study, FT:CT demonstrated statistically significant reductions to baseline at only 5 out of 30 (17 %) time points. The only

comparison to these findings, with the extent of data collection similar to the present study, is from Australian Rules football (ARF), whereby Cormack et al. (2008c) assessed CMJ FT:CT as well as two hormone markers across a season. Their analysis revealed that FT:CT was substantially lower than baseline at 12 out of 20 (60 %) of time points, with one time point in particular showing a substantial decrease of -17.1 ± 21.8 % (ES ± 90 % CI change, -0.77 ± 0.81). In the present study, the most substantial decrease in FT:CT was noted at week 38 (-12.4 %), a two game week towards the end of the competitive season involving long travel for an away fixture. This particular time point also demonstrated reductions in height, FT, AP, TV and increases in CT. No change was noted in MF or PP. These alterations may reflect fatigue manifested in an altered movement strategy whereby countermovement range and speed are changed in order to achieve/maintain peak values (e.g. MF and PP). The greater variability in neuromuscular strategy may therefore be a direct consequence of a propensity to maintain output. Nonetheless, the alterations in movement strategy outputs may have a stronger association to that of increased injury risk. Research has suggested that increased levels of NF may lead to increased electromechanical delay in muscular contraction and reduced rate of force development, that in-turn can predispose to ligamentous injuries such as anterior cruciate ligament ruptures (Krosshaug et al., 2007; Minshull et al., 2012). The possible link between changes in movement strategy and injury incidence were not examined in this study.

Over the course of the competitive season there were substantial increases in MF, whereby 6 out of 30 (20 %) of time points were statistically higher than baseline. The majority of these increases were observed during the middle of the competitive season. These observations in MF would indicate that the players were adapting to training and match loads, as the season progressed. The body has the capacity to adapt to stressors, if

periods of accumulated fatigue from match and training can be reduced to allow positive adaptations to occur (Selye, 1956). Furthermore, lack of observed changes in PP and AF suggest that performance can be maintained even when fatigued through modification of movement strategy (Knicker et al., 2011). This notion is supported in the present study by decreases in TV where players displayed an inability to take-off as quickly during the CMJ in comparison to baseline (12 out of 30 time points, [-3.0 to -6.0 %, $P < 0.05$]), thus taking longer to generate the required force for propulsion from the force platform. Ultimately, if players are taking longer to achieve peak performance outputs this could be the marginal difference in out-manoeuvring an opposing player to allow a goal scoring opportunity that may yield a competitive advantage.

As was observed in Chapters 4 and 5, and in previous research, CMJ height, FT, FT:CT, AP and TV all demonstrated a clear pattern of depression following 90-min of elite competitive football. Therefore, it is reasonable to assume that substantial decrements observed in this study are indicative of low-lowel NF. Although these long-term observations provide indications that these commonly collected measures align to signs of NF, the increases in MF (a gross indicator of performance output) are a direct contrast to our concern regarding decrements in performance. The repeated and routine testing of the CMJ over the course of the season should allow these athletes to execute this movement with well-rehearsed motor-patterns that may contribute to maintenance of peak performance outputs.

To remain cohesive throughout the thesis I continued to monitor hydration status over the season. Testing revealed that Uosm measurers were substantially higher than baseline at 10 out of 33 (30 %) time points. The greatest increase was observed at week 26 (596 mOsmols, +131 %). However, the urine osmolality levels are below the accepted

euhydration cut-off of < 700 (Cheuvront and Sawka, 2005) suggesting that the players were adequately hydrated despite the statistically significant increase to baseline. In support of these findings, the observed 95 % reference range for Uosm (137 to 675 mOsmols) was also below the accepted euhydration cut-off. Therefore, even though statistically significant changes to baseline measures were observed, it is unlikely that the observed changes would be significant enough to affect performance output (Sawka et al., 2007). Although Uosm has shown to be a reliable measure of acute body water loss the results from the present suggest that observing weekly measures may not provide the insight necessary to see the impact of a competitive football season on player hydration status (Armstrong et al., 1994; Oppliger et al., 2005; Kilding et al., 2009; Tam et al., 2011).

6.5: Conclusion

Elite senior football players demonstrated significant fluctuations in both physiological and physical performance markers across a competitive season when compared to baseline measures. The CMJ performance outputs (MF, AF, PP) can be increased and/or maintained over a season despite alterations in jump strategy that may be indicative of low-level NF. Creatine kinase concentrations displayed large increases to baseline, but minimal fluctuation over the season. The collection of only one blood biomarker may not provide the sensitivity necessary to detect signs of muscular or physiological stress in this sub-group. Lastly, the monitoring of player hydration status, via Uosm, demonstrated significant fluctuations to baseline measures over a competitive season but players were able to maintain a euhydrated state that would be unlikely to negatively affect performance output.

Chapter 7: Associations between training/match load and conventional markers of fatigue in elite English Football League Championship players over a competitive season.

7.1: Introduction

In elite football, competition frequency places a heavy influence on the in-season micro-cycle, which typically lasts three to seven days. This micro-cycle will encompass fluctuations in training volume and intensity to allow players to perform at a high level on match-days (Bangsbo et al., 2006). While designing structured training programmes appears to be the first step in performance management, the second most important step towards maximising an adaptive response is monitoring the impact of training/games on players' physiological responses. Adjustments can then be made accordingly to training load and/or recovery strategies during congested periods to maintain peak performance throughout the competitive season and avoid injuries associated with high training workloads (Nimmo and Ekblom, 2007; Gabbett and Jenkins, 2011; Ekstrand et al., 2013; Hulin et al., 2016). Therefore, it is important for practitioners to use markers of fatigue that show meaningful expression of change in relation to fatigability, adaptability, and response to training and game demands (Meeusen et al., 2013).

Chapters 4 and 5 of this thesis showed physical performance and physiological status 48-h post 90-min of competitive football in elite players to be impaired. These findings established that CK testing and CMJ measures show particular promise as acute, simple, non-invasive assessments for monitoring the recovery-fatigue status of elite players. However, observing fatigue markers over short time periods (e.g. post-match), only allows for the observation of the acute/short-term debilitating effects of exercise-induced fatigue. Nevertheless, fatigue can manifest over longer periods of exposure as opposed to short blocks of training/competition as is usually reported in literature (Faude et al., 2011; Rowell et al., 2018).

Physiological adaptation to exercise stress represents the culmination of repeated daily applications of training and match load (Pyne et al., 2009). Thus, the level of fatigue experienced by an athlete is likely the load accumulated from a number of consecutive days. As established in Chapter 6, certain CMJ variables are sensitive to changes in alterations in cumulative load over a season. In addition, it was also found that CK concentrations demonstrated significant increases during the first half of the season. Whether CMJ and CK are sensitive markers of fatigue to weekly training/match load over a season in elite players has yet to be established (Buchheit et al., 2013; Gastin et al., 2013; Thorpe et al., 2015). Therefore, the aim of this chapter was to determine the relationship between cumulative and preceding markers of training/match load and conventional markers of fatigue over a competitive season in elite football players.

7.2: Methods

Participants

Sixteen first-team professional male football players were recruited for this study as described in Chapter 3 (section 3.1). Inclusion criteria included only players who had achieved 20 or more competitive league appearances over the course of the season to observe the physiological and physical effects of those who were playing regularly and available for selection (as described in Chapter 6, Table 6.1).

Experimental Design

All data were collected as described in Chapter 3 (section 3.2). For the purpose of this investigation, only the recovery-fatigue and physical performance markers collected on a Friday (MD -1, 24-h pre-match) were used for subsequent analysis. Individual player training and match external load was monitored throughout the whole season.

Hydration Testing

Hydration testing was measured as described in Chapter 3 (section 3.3.1).

Creatine Kinase Testing

Creatine Kinase testing was measured as described in Chapter 3 (section 3.3.2).

Physical Performance Testing – Counter-movement Jump

The CMJ (a vertical jump test) was performed using a portable force platform (HUR Labs Force Platform 3.8.0.2, Kokkola, Finland) as described in Chapter 3 (section 3.3.3)

Load Monitoring

Global Positioning System

Data was collected using a portable 10-Hz GPS device (Catapault system Minimax S4; Sprint 5.1.7, Catapult Sports, Melbourne, Australia) as described in Chapter 3 (section 3.4.1). To quantify cumulative and preceding training load output, the GPS parameters were totalled for the current week leading up to MD -1 (e.g. Monday to Thursday) and for the previous week (Monday to Friday), for each player. The current weeks GPS training output measures did not include Friday as testing was completed prior to the outdoor training session as described in Chapter 3 (section 3.2).

Match Load

Given the Fédération Internationale de Football Association (FIFA) restrictions at the time of the investigation for the use of tracking devices during match-play, match load was represented by minutes played per player in mid-week fixtures (if present) and/or the previous Saturday leading up to MD -1. Cumulative match load was represented by the adding-up of match-min played across the season.

Statistical Analysis

In order to standardise CK, CMJ and Hydration scores while controlling for player differences in their average levels and variability over the duration of the season, the raw scores were transformed into Z-scores using the following formula:

Z-score = (individual player score - individual players average)/individual players standard deviation

A Z-score represents the number of standard deviations that the response is above or below the mean of the distribution. These Z-scores were the dependent variables (DVs) in our study.

Due to the high multicollinearity between the various independent variables (IVs) from the training load metrics in our study, a factor analysis was undertaken to extract and consolidate common information with factor rotation used to increase the interpretability of the factors. Three factors have been extracted using the Joliffe criterion of retaining factors with eigenvalues > 0.7. The factor analysis has been conducted using Varimax factor rotation to create a simpler structure that is easier to interpret with more factor loadings being either very high (> 0.8) or very low (< 0.2). These three training load factors replaced the original IVs (Table 7.1).

Lastly, ordinary least squares (OLS) multiple regression analysis was undertaken to quantify the relationship between the DVs and the IVs. In addition to the training load IVs, five control IVs were also included. To control for the effects of a mid-week fixture, a binary variable (= 1 if midweek fixture, = 0 if not) was included in the regression analysis. Match load was accounted for by including match min played mid-week, match min played the previous Saturday and cumulative match min over the season. A linear time trend variable was also included using the week number of the season.

Two basic regression models, including the five control IVs, were estimated for each DV. Model I used training min in the current and previous weeks as IVs to model training loads. Model II used the three training load factors as IVs. The level of statistical significance was set at $P < 0.05$ for all t-tests and F-tests. For those where $P < 0.10$ but > 0.05 were referred to as a ‘trend’’ or ‘‘marginally significant’’.

Table 7.1. The new IVs Factor 1, 2 and 3 that were extracted using the Joliffe criterion of retaining factors, values in **bold** indicate factors with eigenvalues > 0.7.

	Factor 1	Factor 2	Factor 3
Training time (min)	**0.8724**	0.0453	-0.3983
TD	**0.8167**	-0.1255	-0.4743
THID	0.4246	-0.2844	**-0.8385**
TSD	0.3606	0.1565	**-0.8049**
HSR	0.4093	-0.3935	**-0.7256**
PL	**0.9010**	-0.2287	-0.2605
PL/min	0.1662	**-0.9099**	0.1002
M/min	0.0360	**-0.8637**	-0.3205
H.I Accels	**0.8156**	0.1028	-0.2562
H.I Decels	**0.9151**	-0.1799	-0.1528
M.I Accels	**0.8814**	-0.2313	-0.3512
M.I Decels	**0.8765**	-0.2237	-0.3640

7.3: Results

Table 7.1 displays the three factors extracted from the factor analysis. Factor 1 picks up the effects of training time (min), TD and acceleration/deceleration focused training outputs. Factor 2 picks up intensity metrics of m/min and PL/min. Lastly, factor 3 picks up distances covered at higher speeds over 5.5 m/s. Table 7.2 shows the descriptive statistics for the recovery-fatigue and physical performance measures, prior to transformation into Z-scores, from every Friday (MD -1) data collection time-point. The training performance data are presented in Table 7.3. The mean total duration of weekly training for the 'current' week (Mon to Thurs) was 155 ± 77 min, with a TD covered from all training sessions of 11309 ± 6256 m and a mean PL of 1144 ± 600 AU. In comparison, the total duration from the 'previous' week (Mon to Fri) was 183 ± 94 min, with a TD covered from all training sessions of 13046 ± 7166 m and a mean PL of 1351 ± 751 AU.

Table 7.4.a to 7.4.k shows the OLS regression models reporting the impact of preceding/ cumulative training and match load on the recovery-fatigue and physical performance measures. The results demonstrated that 4 out of 11 regressions displayed significant coefficients ($P < 0.05$). The regression analysis for Uosm showed Model 1 explained 8 % of variance and Model 2 explained 14 % of variance in Uosm (R^2 = 0.08 and 0.14, respectively). In both models, the time trend variable 'match week' of the season displayed significant positive coefficients. Model 2 also displayed a positive coefficient for Factor 3 (previous week).

Regression analysis for CK showed Model 1 explained 14 % of variance in CK concentrations and Model 2 explained 18 % of variance (R^2 = 0.14 and 0.18, respectively). Cumulative min across the season displayed significant positive

coefficients in both models (Table 7.4.b). Training min (previous week) also displayed a significant positive coefficient in Model 1 and similarly Factor 1 (previous week) displayed a significant positive coefficient, which as described previously picked up the effect of training min in its factor.

CMJ AF and AP displayed significant coefficients from the regression analyses. Regression analysis for CMJ AF showed Model 1 explained 10 % variance and Model 2 explained 12 % of variance in CMJ AF output (R^2 = 0.10 and 0.12, respectively). The time trend variable 'match week' of the season displayed significant positive coefficients in both models (Table 7.4.i). Regression analysis for CMJ AP displayed a significant negative coefficient for Factor 3 (previous week) in Model 2, but overall the model explained only 6 % of variance in CMJ AP output (Table 7.4.j).

Further, the results demonstrated that 7 out of the 11 regressions had no significant coefficients for either Model 1 or Model 2 ($P > 0.05$). However, trends were indicated in CMJ CT regression analysis in Model 1 for positive coefficients of 'mid-week' and training min (Table 7.4.d). Regression analysis revealed trends for CMJ FT in Model 1 for min-previous sat (Table 7.4.e) and CMJ FT:CT in Model 1 and 2 for 'mid-week (Table 7.5.f).

Table 7.2. Descriptive statistics, medians (interquartile ranges) for CMJ, CK and Uosm from the entire season.

	Mean ± SD	Median (IQR)
Uosm (mOsmols)	371 ± 182	330 (240 to 470)
CK (u/l)	448 ± 328	349 (231 to 566)
CMJ height (cm)	35.3 ± 4.8	34.6 (32 to 38)
CMJ CT (ms)	838.7 ± 123.9	835.8 (773 to 895)
CMJ FT (ms)	554.4 ± 49.9	552.5 (525 to 580)
CMJ FT:CT	0.68 ± 0.14	0.67 (0.61 to 0.73)
CMJ MF (N)	1899.5 ± 253.2	1858.5 (1751 to 1979)
CMJ PP (W)	4129.2 ± 579.5	3988.5 (3795 to 4311)
CMJ AF (N)	1056.1 ± 92.3	1047.2 (991 to 1085)
CMJ AP (W)	1077.4 ± 183.1	1054.8 (946 to 1197)
CMJ TV (m/s)	2.62 ± 0.16	2.61 (2.51 to 2.73)

Table 7.3. Load and intensity measures for training during the current week (Mon to Thurs) and the previous week (Mon to Fri). Data are presented as mean (± SD).

	Training time (min)	TD (m)	HID (m)	SD (m)	HSR (m)	PL (au)	PL/min ($au.min^{-1}$)	M/min ($m.min^{-1}$)	H.I Acc (n)	H.I Decel (n)	Med.Acc (n)	Med.Dec (n)
Current week (Mon to Thurs)	155 (77)	11309 (6256)	969 (755)	142 (166)	787 (620)	1144 (600)	8 (2)	75 (16)	295 (169)	116 (67)	744 (377)	644 (321)
Previous week (Mon to Fri)	183 (94)	13046 (7166)	1043 (866)	153 (177)	870 (726)	1351 (751)	8 (2)	73 (14)	361 (211)	138 (80)	885 (493)	764 (414)

Table 7.4.a OLS regression analysis of preceding and cumulative training/match load on Uosm over a season.

Dependent Variable: Uosm	**Model 1**	**Model 2**
Constant	-0.2093 (0.2905)	*-0.2986 (0.1730)*
Match week	**0.0263 (0.0082)**	**0.0237 (0.0097)**
Mid-week	-0.0830 (0.2024)	0.2828 (0.2343)
Min-previous Sat	-0.0003 (0.0018)	-0.0008 (0.0020)
Min mid-week	-0.0017 (0.0030)	-0.0047 (0.0036)
Cumulative min	-0.0002 (0.0001)	-0.0001 (0.0001)
Training min	-9.42562e-005 (0.0010)	
Training min - previous week	-0.0005 (0.0007)	
Factor 1		0.0073 (0.0766)
Factor 2		0.0803 (0.0712)
Factor 3		0.0093 (0.0668)
Factor 1 – Previous week		-0.0203 (0.0681)
Factor 2 – Previous week		0.0454 (0.0717)
Factor 3 – Previous week		**0.1657 (0.0641)**
Goodness of fit		
s	0.8308	0.8175
R^2	0.0819	0.1364
F	**0.018***	**0.010****

Statistical significance (P < 0.05) is indicated in **bold**, and a trend (where P < 0.10 but < 0.05) is indicated in *italics.*

Standard errors in parentheses (two-tailed test). s = standard error of regression; F = test of overall significance of regression (F one-tailed test).

Table 7.4.b. OLS regression analysis of preceding and cumulative training/match load on CK over a season.

Dependent Variable: CK	**Model 1**	**Model 2**
Constant	-1.3158 (0.5393)	-0.2297 (0.2633)
Match week	-0.0193 (0.0174)	-0.0141 (0.0180)
Mid-week	*0.7648 (0.4082)*	0.6175 (0.4198)
Min-previous Sat	-0.0004 (0.0028)	-0.0004 (0.0030)
Min mid-week	-0.0056 (0.0056)	-0.0036 (0.0056)
Cumulative min	**0.0007 (0.0003)**	**0.0006 (0.0003)**
Training min	*0.0031 (0.0017)*	
Training min - previous week	**0.0035 (0.0013)**	
Factor 1		0.1103 (0.1273)
Factor 2		*0.2245 (0.1300)*
Factor 3		-0.0765 (0.1201)
Factor 1 – Previous week		**0.2359 (0.1152)**
Factor 2 – Previous week		-0.2140 (0.1353)
Factor 3 – Previous week		-0.1568 (0.0945)
Goodness of fit		
s	0.9990	0.9964
R^2	0.1387	0.1814
F	*0.055*	*0.079*

Statistical significance (P < 0.05) is indicated in **bold**, and a trend (where P < 0.10 but < 0.05) is indicated in *italics.*

Standard errors in parentheses (two-tailed test). s = standard error of regression; F = test of overall significance of regression (F one-tailed test).

Table 7.4.c. OLS regression analysis of preceding and cumulative training/match load on CMJ height over a season.

Dependent Variable: CMJ Height	**Model 1**	**Model 2**
Constant	0.3252 (0.3717)	0.3164 (0.2023)
Match week	-0.0112 (0.0104)	-0.0100 (0.0109)
Mid-week	-0.0414 (0.2131)	-0.0470 (0.2212)
Min-previous Sat	-0.0020 (0.002175)	-0.0017 (0.0023)
Min mid-week	0.0021 (0.0035)	0.0017 (0.0036)
Cumulative min	-0.0001 (0.0002)	-0.0001 (0.0002)
Training min	0.0002 (0.0012)	
Training min - previous week	-6.05688e-005 (0.0009)	
Factor 1		0.0137 (0.0900)
Factor 2		0.0093 (0.0789)
Factor 3		-0.0070 (0.0791)
Factor 1 – Previous week		-0.0117 (0.0783)
Factor 2 – Previous week		-0.0107 (0.0803)
Factor 3 – Previous week		-0.0643 (0.0773)
Goodness of fit		
s	0.973	0.9818
R^2	0.0527	0.0568
F	0.187	0.467

Statistical significance ($P < 0.05$) is indicated in **bold**, and a trend (where $P < 0.10$ but < 0.05) is indicated in *italics.*

Standard errors in parentheses (two-tailed test). s = standard error of regression; F = test of overall significance of regression (F one-tailed test).

Table 7.4.d. OLS regression analysis of preceding and cumulative training/match load on CMJ CT over a season.

Dependent Variable: **CMJ CT**	**Model 1**	**Model 2**
Constant	-0.4124 (0.3669)	*-0.3445 (0.2024)*
Match week	0.0081 (0.0102)	0.0053 (0.0107)
Mid-week	*0.3546 (0.2087)*	0.3495 (0.2171)
Min-previous Sat	0.0013 (0.0021)	0.0010 (0.0023)
Min mid-week	0.0030 (0.0034)	0.0034 (0.0036)
Cumulative min	-1.29019e-005 (0.0002) *0.0019 (0.0011)*	1.49248e-005 (0.0002)
Training min	-0.0014 (0.0009)	
Training min - previous week		
Factor 1		0.1132 (0.0895)
Factor 2		-0.0204 (0.0824)
Factor 3		-0.0416 (0.0780)
Factor 1 – Previous week		-0.1315 (0.0799)
Factor 2 – Previous week		-0.0279 (0.0795)
Factor 3 – Previous week		0.0984 (0.0791)
Goodness of fit		
s	0.9480	0.9582
R^2	0.0512	0.0523
F	0.216	0.562

Statistical significance ($P < 0.05$) is indicated in **bold**, and a trend (where $P > 0.05$ but $P < 0.10$) is indicated in *italics.*

Standard errors in parentheses (two-tailed test). s = standard error of regression; F = test of overall significance of regression (F one-tailed test).

Table 7.4.e. OLS regression analysis of preceding and cumulative training/match load on CMJ FT over a season.

Dependent Variable: CMJ FT	**Model 1**	**Model 2**
Constant	*0.6611 (0.3671)*	*0.4914 (0.2041)*
Match week	-0.0152 (0.0103)	-0.0163 (0.0108)
Mid-week	-0.1886 (0.2121)	-0.1936 (0.2201)
Min-previous Sat	*-0.0038 (0.0021)*	-0.0034 (0.0023)
Min mid-week	0.0017 (0.0034)	0.0020 (0.0036)
Cumulative min	-2.43230e-006 (0.0002)	1.05681e-005 (0.0002)
Training min	-0.0010 (0.0011)	
Training min - previous week	5.50298e-006 (0.0009)	
Factor 1		-0.0032 (0.0907)
Factor 2		-0.0566 (0.0830)
Factor 3		0.0510 (0.0783)
Factor 1 – Previous week		-0.0066 (0.0799)
Factor 2 – Previous week		0.0333 (0.0787)
Factor 3 – Previous week		-0.0220 (0.0796)
Goodness of fit		
s	0.9581	0.9680
R^2	0.0517	0.0536
F	0.211	0.541

Statistical significance ($P < 0.05$) is indicated in **bold**, and a trend (where $P < 0.10$ but < 0.05) is indicated in *italics.*

Standard errors in parentheses (two-tailed test). s = standard error of regression; F = test of overall significance of regression (F one-tailed test).

Table 7.4.f. OLS regression analysis of preceding and cumulative training/match load on CMJ FT:CT over a season.

Dependent Variable: CMJ FT:CT	**Model 1**	**Model 2**
Constant	0.4871 (0.3615)	*0.3931 (0.2017)*
Match week	-0.0071 (0.0102)	-0.0044 (0.0107)
Mid-week	*-0.3849 (0.2102)*	*-0.3851 (0.2180)*
Min-previous Sat	-0.0008 (0.0021)	-0.0003 (0.0023)
Min mid-week	-0.0018 (0.0034)	-0.0024 (0.0035)
Cumulative min	-6.57141e-005 (0.0002)	-8.71143e-005 (0.0002)
Training min	-0.0015 (0.0011)	
Training min - previous week	0.0010 (0.0009)	
Factor 1		-0.0644 (0.0896)
Factor 2		0.0199 (0.0828)
Factor 3		0.0539 (0.0785)
Factor 1 – Previous week		0.0925 (0.0789)
Factor 2 – Previous week		0.0310 (0.0793)
Factor 3 – Previous week		-0.1172 (0.0801)
Goodness of fit		
s	0.9560	0.9640
R^2	0.0429	0.0485
F	0.333	0.625

Statistical significance ($P < 0.05$) is indicated in **bold**, and a trend (where $P < 0.10$ but < 0.05) is indicated in *italics.*

Standard errors in parentheses (two-tailed test). s = standard error of regression; F = test of overall significance of regression (F one-tailed test).

Table 7.4.g. OLS regression analysis of preceding and cumulative training/match load on CMJ MF over a season.

Dependent Variable: **CMJ MF**	**Model 1**	**Model 2**
Constant	-0.4173 (0.3850)	-0.1097 (0.2135)
Match week	0.0090 (0.0109)	0.0105 (0.0113)
Mid-week	-0.2312 (0.2239)	-0.2172 (0.2308)
Min-previous Sat	-0.0007 (0.0022)	-0.0007 (0.0024)
Min mid-week	0.0025 (0.0036)	0.0027 (0.0037)
Cumulative min	0.0001 (0.0002)	7.17909e-005 (0.0002)
Training min	0.0005 (0.0012)	
Training min - previous week	0.0012 (0.0009)	
Factor 1		0.0713 (0.0949)
Factor 2		-0.0560 (0.0877)
Factor 3		-0.0654 (0.0831)
Factor 1 – Previous week		0.0915 (0.0835)
Factor 2 – Previous week		0.1154 (0.0840)
Factor 3 – Previous week		-0.0839 (0.0849)
Goodness of fit		
s	1.0181	1.0205
R^2	0.0286	0.0457
F	0.623	0.673

Statistical significance ($P < 0.05$) is indicated in **bold**, and a trend (where $P < 0.10$ but < 0.05) is indicated in *italics.*

Standard errors in parentheses (two-tailed test). s = standard error of regression; F = test of overall significance of regression (F one-tailed test).

Table 7.4.h. OLS regression analysis of preceding and cumulative training/match load on CMJ PP over a season.

Dependent Variable: CMJ PP	**Model 1**	**Model 2**
Constant	-0.2608 (0.3730)	0.0523 (0.2081)
Match week	0.0093 (0.0105)	0.0108 (0.0111)
Mid-week	-0.1090 (0.2184)	-0.1097 (0.2260)
Min-previous Sat	-0.0007 (0.0022)	-0.0012 (0.0024)
Min mid-week	0.0027 (0.0035)	0.0024 (0.0036)
Cumulative min	-0.0002 (0.0002)	-0.0002 (0.0002)
Training min	0.0012 (0.0012)	
Training min - previous week	0.0005 (0.0009)	
Factor 1		0.0548 (0.0927)
Factor 2		0.0052 (0.0849)
Factor 3		-0.0685 (0.0808)
Factor 1 – Previous week		0.0549 (0.0815)
Factor 2 – Previous week		0.0630 (0.0819)
Factor 3 – Previous week		-0.0350 (0.0827)
Goodness of fit		
s	0.9865	0.9954
R^2	0.0267	0.0311
F	0.667	0.892

Statistical significance ($P < 0.05$) is indicated in **bold**, and a trend (where $P < 0.10$ but < 0.05) is indicated in *italics.*

Standard errors in parentheses (two-tailed test). s = standard error of regression; F = test of overall significance of regression (F one-tailed test).

Table 7.4.i. OLS regression analysis of preceding and cumulative training/match load on CMJ AF over a season.

Dependent Variable: CMJ AF	**Model 1**	**Model 2**
Constant	-0.5543 (0.3526)	-0.2848 (0.1949)
Match week	**0.0273 (0.0100)**	**0.0326 (0.0104)**
Mid-week	-0.2663 (0.2063)	-0.2444 (0.2117)
Min-previous Sat	-0.0020 (0.0021)	-0.0022 (0.0022)
Min mid-week	-0.0014 (0.0033)	-0.0024 (0.0034)
Cumulative min	-1.83372e-005 (0.0002)	-8.69517e-005 (0.0002)
Training min	0.0003 (0.0011)	
Training min - previous week	0.0012 (0.0009)	
Factor 1		0.0099 (0.0873)
Factor 2		0.0716 (0.0801)
Factor 3		-0.0473 (0.0759)
Factor 1 – Previous week		0.1010 (0.0763)
Factor 2 – Previous week		0.0636 (0.0767)
Factor 3 – Previous week		-0.1136 (0.0775)
Goodness of fit		
s	0.9315	0.9317
R^2	0.1045	0.1242
F	**0.006****	**0.014***

Statistical significance ($P < 0.05$) is indicated in **bold**, and a trend (where $P < 0.10$ but < 0.05) is indicated in *italics.*

Standard errors in parentheses (two-tailed test). s = standard error of regression; F = test of overall significance of regression (F one-tailed test).

Table 7.4.j. OLS regression analysis of preceding and cumulative training/match load on CMJ AP over a season.

Dependent Variable: CMJ AP	**Model 1**	**Model 2**
Constant	0.0717 (0.3553)	0.1420 (0.1964)
Match week	0.0061 (0.0100)	0.0104 (0.0105)
Mid-week	-0.2856 (0.2075)	-0.2915 (0.2137)
Min-previous Sat	-0.0029 (0.0021)	-0.0029 (0.0022)
Min mid-week	-0.0031 (0.0033)	-0.0042 (0.0034)
Cumulative min	3.23533e-005 (0.0002)	-2.15898e-005 (0.0002)
Training min	-0.0011 (0.0011)	
Training min - previous week	0.0013 (0.0009)	
Factor 1		-0.0779 (0.0873)
Factor 2		0.0577 (0.0801)
Factor 3		0.0540 (0.0757)
Factor 1 – Previous week		0.0784 (0.0772)
Factor 2 – Previous week		0.0304 (0.0773)
Factor 3 – Previous week		**-0.1587 (0.0769)**
Goodness of fit		
s	0.9401	0.9430
R^2	0.0525	0.0679
F	0.198	0.314

Statistical significance ($P < 0.05$) is indicated in **bold**, and a trend (where $P < 0.10$ but < 0.05) is indicated in *italics.*

Standard errors in parentheses (two-tailed test). s = standard error of regression; F = test of overall significance of regression (F one-tailed test).

Table 7.4.k. OLS regression analysis of preceding and cumulative training/match load on CMJ TV over a season.

Dependent Variable: CMJ TV	**Model 1**	**Model 2**
Constant	0.4302 (0.3517)	*0.3772 (0.1960)*
Match week	-0.0157 (0.0099)	-0.0137 (0.0104)
Mid-week	0.0170 (0.2045)	-0.0001 (0.2119)
Min-previous Sat	-0.0026 (0.0021)	-0.0025 (0.0022)
Min mid-week	0.0010 (0.0033)	0.0003 (0.0034)
Cumulative min	-4.92448e-005 (0.0002)	-6.66714e-005 (0.0002)
Training min	-0.0003 (0.0011)	
Training min - previous week	-1.84188e-005 (0.0009)	
Factor 1		-0.0267 (0.0871)
Factor 2		0.0343 (0.0805)
Factor 3		0.0200 (0.0763)
Factor 1 – Previous week		-0.0138 (0.0767)
Factor 2 – Previous week		0.0155 (0.0771)
Factor 3 – Previous week		-0.0760 (0.0779)
Goodness of fit		
s	0.9299	0.9370
R^2	0.0668	0.07362
F	*0.082*	0.244

Statistical significance ($P < 0.05$) is indicated in **bold**, and a trend (where $P < 0.10$ but < 0.05) is indicated in *italics.*

Standard errors in parentheses (two-tailed test). s = standard error of regression; F = test of overall significance of regression (F one-tailed test).

7.4: Discussion

The main findings of the present study indicate that our markers of preceding/cumulative training and match load had minimal or small to no effects on the conventional recovery-fatigue markers used in this study. Creatine kinase concentrations displayed the greatest sensitivity to cumulative match load and Uosm to the time trend variable 'match-week' of the season. Match-week also demonstrated significant associations to increases in CMJ average force (AF). The recovery-fatigue variables used demonstrate poor sensitivity to training load outputs measured via GPS over a season.

In Chapter 6, several CMJ outputs demonstrated significant fluctuations to baseline values during the season. The purpose of the current study was to determine if these fluctuations aligned to training/match demands. For a marker of fatigue to be considered useful in monitoring changes in physical or physiological status, it needs to be capable of reflecting fatigue, but also an improvement brought about by an appropriate training stimulus and/or sensitive enough to detect the impact of interventions to allow recovery (Meeusen et al., 2013).

The present study demonstrated significant associations between the linear time trend variable of 'match-week' and CMJ AF, indicating that as the season progressed so did the mean force produced by players during the CMJ. This change coincided with trends indicating increases in CMJ CT associated with increasing training min and the presence of a mid-week fixture. However, these associations were small and only marginally significant ($P < 0.10$ but $P > 0.05$). There were no observed associations between any training/match load outputs and CMJ height, MF, PP and TV. These findings would imply that these highly trained athletes are able to achieve/maintain peak performance despite minor changes in jump strategy indicative of low-level NF. Contraction time

accounts for three distinct phases of the CMJ; the unweighting phase, the braking phase and propulsion phase (McMahon et al., 2018). Although the analysis did not take into account during which phase time increased, one can assume that players were taking longer to produce maximum force outputs. This perhaps being a subconscious strategy employed to sustain (or maximise) performance under low levels of fatigue.

In support of these findings, previous research has also struggled to establish a relationship between accumulated training load and CMJ outputs for the detection of NF. Thorpe et al. (2017) addressed the sensitivity of potential fatigue measures to daily training load accumulated over 2 to 4 days during a 17-day in-season competitive period in 10 senior outfield players. Their results showed insignificant negligible correlations between variability in CMJ height and THIR distance across all accumulated days, indicating that the sensitivity of CMJ height to changes in training load is not improved when compared with training loads beyond the previous day of training. Nonetheless, they did demonstrate that other indices of morning-measured fatigue such as 'perceived ratings of fatigue' and submaximal heart rate were sensitive to fluctuations in load accumulated over 2 to 4 days.

Furthermore, Rowell et al. (2018) investigated fatigue responses over a competitive season in 23 elite outfield players competing in the A-league. They examined the relationship between internal load (RPE), fatigue measures (CMJ FT:CT, testosterone and cortisol) and match performance (coaches' ratings) over a period of 3 to 28 days of cumulative load. Their results demonstrated a large number of unclear or trivial effects on CMJ FT:CT as a result of changes in training load. It was suggested this may be due to FT:CT being more sensitive to external load (e.g. accelerometer and GPS derived metrics) than RPE based internal load as collected in the study. It should also be noted

that Rowell et al. (2018) conducted CMJ testing on MD -1, similar to the present study, which may have had an influence on the observed relationships due to the specific time point of the micro-cycle. The present study observed marginally significant associations between weeks with a scheduled mid-week fixture and decreases in FT:CT. However, this was more likely associated with increases in CT component as discussed previously. Collectively, these findings indicate that CMJ indices are largely insensitive to preceding and cumulative load when observed over longer periods in elite football players, and recent load may be more important due to the peaks and and-troughs within a short time frame that only lead to acute changes in physiological status (Thorpe, 2017; Chapter 4; Chapter 5). As long as practitioners adhere to structured training regimes, senior players at this elite level are able to maintain physical performance output by MD -1 for a Saturday fixture.

Although observed peak performance outputs from a CMJ can be maintained across a season, there was a weak-positive association between CK concentration and cumulative match-time suggestive of increased signs of muscular stress in response to match-min played across the season in the present study. Further, there seems to be a lagged response in training min and training outputs (Factor 1) from the previous week suggesting chronically elevated CK levels to increasing training time and possibly to high metabolic activities, such as accelerations and decelerations, which have a high eccentric component (as described in Chapter 2.7). Similar associations between CK and cumulative match-min have been previously described in elite Polish football players (Nowakowska et al., 2019). In a longitudinal study, comparable to our own, Nowakowska et al. (2019) undertook repeated blood analyses, 24-h after each match, over an entire competitive season to examine the relationship between routine plasma biomarker levels and recovery efficiency. Creatine kinase concentrations demonstrated

a weak-positive correlation to cumulative match-time in midfielders/defenders. The findings from Nowakowska et al. (2019) and our own study do not support the notion of adaptation to repeated demands (Law of Accommodation) in this cohort as suggested in Chapter 6. However, in the study conducted by Nowakowska et al. (2019) the methodology should be questioned for the time point of blood collection at 17 to 24-h after each competitive match. In a typical football micro-cycle this time-point is usually devoted to recovery and therefore would not influence subsequent training load modification. Further, at this time-point the expected profile of response for CK is higher in comparison to 48-h post-match and may contribute to the association to cumulative match-time shown (Coelho et al., 2011; Nedelec et al., 2014; de Hoyo et al., 2016).

Preceding and cumulative training/match load explained a low proportion of variability in Uosm levels. However, there were significant associations to the time trend variable 'match-week', suggesting that as the season progressed players' average hydration score increased moving towards a more hypohydrated state. The results suggest that for every week over the season there was a 0.02 shift in the player's deviation from their absolute average (Z-score). The significance of this per-week is relatively small but over season may lead to detrimental performance changes if not able to maintain a 'euhydrated' state. Although the validity of Uosm has been questioned in previous chapters, it shows tendency to be sensitive to cumulative load. However, the interpretation of Uosm needs to take into consideration the threshold of 'euhydration' previously reported (Chapter 6) to help determine if these individual shifts in hydration status are of clinical importance.

In a population with selection bias, these players were accustomed to the regular demands of football match-play and made a minimum of 22 competitive league appearances during the season. Although these players did encounter injuries, this data was omitted

from the analysis and in general this cohort were available for selection for between 28 and 49 matches over the season. In this bias population, the findings demonstrated that fixture congestion had minimal influence on the players' physical/physiological response. The presence of a mid-week fixture showed weak positive trends in increased CMJ CT and CK concentration, suggesting that players are adequately recovered by MD -1 despite the presence of a mid-week fixture. In a recent investigation, Owen et al. (2019) found CK concentrations to be significantly higher at MD +2 in a congested period (2 consecutive games in 4-days or less) vs. a non-congested period (2 consecutive games within 5-days or more) in 23 elite male European football players (ES: 0.27, $P < 0.05$). However, the post-match CK concentrations were relatively low during both congested vs. non-congested periods (206 ± 116 and 176 ± 111 u/l, respectively), suggesting that the physical demand of match-play did not represent a much higher muscular stimulus than the usual training load of these players. This supports the notion that elite football players are accustomed to the rigour demands of competitive match-play and are potentially able to recover post-match play with greater efficiency.

Taking into consideration the bias in this cohort of elite footballers who were playing regularly (with injury data omitted from this investigation), a case study was undertaken on several players who sustained a non-contact injury during the season (Appendix 1). This was undertaken to see whether any trends could be observed in the data prior to injury incidence, such as decrements in physical performance or increases in CK concentration. One player in this investigation whom had 22 league appearances, showed significant deviations from baseline measures and Z-score changes in Uosm and CK (+53.2 and 587 %; Z-score 1.3 and 1.9, respectively). Further, significant deviations from baseline measures in CMJ height, CT, FT, FT:CT and TV (-4.8 to -12.9 %) prior to injury incidence were observed, but Z-score change was minimal. Conversely, a different player

whom achieved 33 league appearances demonstrated no significant changes in any of the fatigue markers prior to injury incidence and showed some improvements in CMJ physical performance. Overall, the data displayed no pattern or trend prior to injury incidence and suggested that these markers are not consistently sensitive to fatigue induced changes between individuals that may lead to injury.

The previous chapters of this thesis have questioned the validity of observing a single biomarker such as CK due to its high inter-individual variability. Researchers have agreed that multiple markers should be measured together when attempting to detect over-training and/or fatigue (Lee et al., 2017). Alongside CK, Owen et al. (2019) observed salivary cortisol and IgA but failed to observe any significant changes in these markers in response to fixture congestion. The present study aimed to address the high inter-individual variability by reporting the recovery-fatigue variables as 'Z-scores', representing the variance in score in comparison to an individual's average across the season. This method for analyses could be a factor in determining the potential for observed effects. Malone et al. (2018) calculated individual Z-scores for analysis between pre-training CK and CMJ height and subsequent training intensity output. Their analyses revealed that CK Z-scores of +1 and CMJ Z-scores of -1 related to reductions in THIR outputs, explosiveness and acceleration/deceleration outputs during training. Utilising Z-scores in the field can allow the chosen analyses to account for individual differences in physical and physiological response.

This study supports the importance of structured training regimes (micro-cycles) with days devoted to recovery to allow players to restore physiological homeostasis. The literature shows that fixture congestion and insufficient recovery over extended periods may contribute to potentially long-term debilitating effects associated with overtraining,

such as increased risk of injury or illness (Nimmo and Ekblom, 2007; Ekstrand et al., 2013; Hulin et al., 2014). If markers of recovery-fatigue are not sensitive to changes in preceding and cumulative training load, then the validity of their use is questioned for detecting over-training and when a player may be at increased risk of injury. Research in elite football has further provided evidence that physical and technical performance (skill-related) is maintained throughout a season even when congested periods are prolonged (Djaoui et al., 2014; Carling et al., 2015).

The findings from this investigation revealed that preceding and cumulative match/training load explained a low proportion of variance for all recovery-fatigue and physical performance outputs used (8 to 18 %, $P < 0.05$). However, the study was able to establish weak significant coefficients between some measures of match/training load and these measures. Perhaps, the measures used are not sensitive enough for this particular cohort to detect signs of cumulative fatigue from training/ games. However, time restraints due to the quick turnaround in fixtures restrict the use of more invasive tests, and maximal performance tests after the game may further debilitate the physical status of players and/or increase the risk of injury (Carling et al., 2015).

7.5: Conclusion

The conventional recovery-fatigue markers used in this study are relatively insensitive to preceding/cumulative physical training load outputs. Creatine kinase, Uosm, CMJ CT and FT:CT may display some sensitivity to preceding/cumulative match load and time-trend across the season. However, elite senior football players can produce increases in AF through small alterations in the time taken to produce the CMJ movement. In general, players display the ability to be adequately recovered by MD -1 despite the imposition of a mid-week fixture, or culmination of training/match load.

Chapter 8: Synthesis of Findings

8.1: Synthesis of Findings

The studies performed in this thesis provided novel data in regard to fatigue monitoring in elite football players over the course of a season. The aim of this chapter is to interpret and integrate the findings obtained within this thesis. The realisation of the aims of the thesis will be confirmed in relation to the original aims and objectives presented in Chapter 1. The possible applications and limitations will be discussed.

The specific aims of this thesis were:

1. To assess the acute changes in physiological and physical performance markers, from multiple games across a season, after completing 90-min of competitive football in elite players 24-h pre to 48-h post-match.

2. To analyse the sensitivity of physiological and physical performance markers to preceding match-load from multiple competitive matches across a season, in elite players.

3. To examine the longitudinal fluctuations in physiological and physical performance markers across a season in elite players.

4. To examine the association between markers of training/match load and physiological and physical performance markers over a football season to determine the sensitivity of these markers to preceding and cumulative load.

8.2: Achievement of aims and objectives

Acute changes in physiological and physical performance markers after completing 90-min of competitive football in elite senior level football players:

Aim 1 was addressed in Chapter 4, through the assessment of the acute changes in physiological and physical performance markers, across a season, after 90-min of competitive football. The intention of this chapter was to establish and interpret three commonly collected markers (Uosm, CK concentration and CMJ height) in a cohort of 18 elite players. These markers were deemed practical, time efficient and easy to implement in an elite sporting environment for the regular assessment of large groups and have been previously used within similar settings.

The main findings of Chapter 4 were that after 90-min of competitive football, CMJ height collected via a 'Just-Jump System' was able to detect an alteration in physical performance in the direction expected at 48-h post-match (ES: -0.37, CI = 0.47 to 3.79). Jump height displayed a small percentage change in comparison to previously reported research (3.9 % vs 6.1 % to 7.5 %; Nedelec et al., 2014; de Hoyo et al., 2016). However, this was attributed to differences in testing procedures, equipment used, the time-point of data collection and the population assessed. Creatine kinase concentrations, from capillary blood, significantly increased and seems to be sensitive to the overall demands of match-play that may be associated with 90-min of competitive football. Nevertheless, the large reference range of CK concentration in male athletes (82 to 1083 u/l) suggests minimal muscular or fatigue related stress was present after 90-min of elite competitive football (Mougios, 2007). Lastly, player hydration status measured via Uosm from a handheld urinary refractometer (osmocheck) displayed no significant change pre- to post-match and reflects either that elite players are able to achieve rehydration by 48-h

post-game, or the equipment employed within the limitations of the research environment was not sensitive to changes.

A limitation of this chapter was the exclusion of physical match performance outputs and their association to the recovery-fatigue markers used. Further, due to the inconsistency in literature regarding the validity of CMJ height to detect NF, combined with the small post-match changes reported in this chapter, a force platform was used for subsequent research to allow an advanced assessment of physical performance.

Sensitivity of physiological and physical performance markers to preceding match-load:

Chapter 5 mirrored that of Chapter 4 but built upon the previous findings to address **aim 2** of the thesis by determining the sensitivity of physiological and physical performance markers to preceding match external load in a sub-group of 18 elite players. Recent research has suggested that concentric focused outputs from the push-off phase, such as CMJ height, lacks the resolution to detect NF in elite athletes (Balloch, 2018). Therefore, this chapter explored additional variables collected through CMJ testing using a force platform (CT, FT, FT:CT, PP, MF, AF, AP and TV).

The main findings of Chapter 5 were that CK concentrations at 48-h post-match do not seem to be linked to match external load but are sensitive to the overall demands of 90-min of competitive football (supporting findings from Chapter 4). Further, CMJ variables related to both performance (FT) and movement strategy (FT:CT, AP) are sensitive to changes in NF 48-h post competitive football. The CMJ output measures of FT and AP displayed the most sensitivity to fatigue at 48-h post-match (ES = -0.45 to -0.63) with moderate associations to most physical match performance variables (HID, TSD, HIN,

TSN, EXS, MAcc and MDec) completed during 90-min of competitive match-play (r = -0.40 to -0.50). Jump height displayed no significant difference 24-h pre to 48-h post-match and no sensitivity to match external load, however a small effect size and narrow CIs were reported post-match (ES: -0.35, CI = -3.1 to 0.01 cm), which provides a better method for assessing real-world change in jump performance. Similar to the findings of Chapter 4, Uosm did not reveal any significant difference at 48-h post-match and were below the accepted euhydration cut-off set at < 700 mOsmols (Cheuvront and Sawka, 2005).

The findings of Chapter 4 and Chapter 5 demonstrated that CK concentrations and CMJ height, FT, FT:CT, AP and TV are acutely affected 48-h following 90-min of competitive match-play in elite players. These chapters addressed previous issues in research regarding 'one-off' data sets and specific sub-groups of elite players. The observations from these experimental chapters took place across two seasons in the EFL Championship and displayed high ecological validity demonstrating real-world changes in physical and physiological status in elite players. Further, these chapters highlighted the position specific responses in these markers and the individual differences from 90-mins of competitive football.

Longitudinal analyses of physiological and physical performance markers across a season in the EFL:

After establishing and quantifying the acute changes in these conventional markers of fatigue 48-h post competitive match-play used in Chapter 4 and Chapter 5, Chapter 6 was designed to address **aim 3** of this thesis by investigating the longitudinal alterations in these markers to detect signs of cumulative fatigue across a season. A total of 16 elite senior level football players were used in this analysis, all having achieved 20 or more

appearances over the season. To the author's knowledge, this was the first study to address weekly variations in these measures over a competitive season. The main findings of Chapter 6 were that CK concentrations demonstrated large increases to baseline measures but limited fluctuation over the season (61.7 to 158.7 %, $P < 0.05$), and CMJ max force can be increased (5.1 to 7.0 %, $P < 0.05$) despite fluctuations in CMJ movement strategy focused outputs (TV, FT:CT and AP) that may be indicative of NF. Hydration measured via Uosm showed evident fluctuations to baseline measures, however it was unlikely that the observed changes would be significant enough to induce detrimental performance changes (Sawka et al., 2007).

This study was able to establish fluctuations in these conventional markers of fatigue across a season but was not able to conclude on the sensitivity of them to preceding and cumulative match/training demands. Data in this chapter is novel and provides 95 % reference ranges and normative values for this sub-group of elite players to aid future interpretation of meaningful changes in physiological and physical performance measures.

Association between markers of training/match load and physiological and physical performance markers over a football season:

Aim 4 was addressed in Chapter 7, to assess all possible limitations of previous chapters and examined the association between the recovery-fatigue variables used in Chapter 4, 5 and 6 to preceding and cumulative match/training load over a season. The high inter-individual variability in the physiological and physical performance variables was addressed by transforming raw scores into 'Z-scores' for the analysis. The same cohort from chapter 6 were included in this study but only the recovery-fatigue variables from a Friday (MD -1) were used for analysis. Preceding and cumulative training load was

quantified by incorporating weekly training output totals from the week leading up to the Friday testing (Mon-Thurs) and the week prior (Mon to Fri). The observation of total weekly load has been used for monitoring acute spikes in training load in comparison to chronic load over the preceding weeks (Banister and Calvert, 1980; Hulin, 2016). The rationale of observing cumulative load per week ties in appropriately with the micro-cycles used in elite football, which typically lasts three to seven days in duration. Match load was quantified by including match-min completed mid-week (if present) and the previous Saturday. Whereas match cumulative load was quantified by the accumulation of match-min played across the season.

The main findings from Chapter 7 were that CK concentrations displayed the greatest sensitivity to cumulative match load and Uosm to the time trend variable 'match-week' of the season. Further, 'match-week' also demonstrated significant associations to increases in CMJ AF. The recovery-fatigue variables used demonstrated minimal association to training load outputs measured via GPS over a season. The results suggested a trend for increasing CMJ CT to increasing training minutes and the presence of mid-week fixture, but these associations were small and only marginally significant ($P > 0.5$ but $P < 0.10$). These findings are in support of Chapter 6 suggesting highly trained athletes are able achieve/maintain peak performance despite minor changes in jump strategy indicative of low-level NF. Thus, as long as practitioners adhere to structured training regimes allowing for sufficient recovery, senior players at this elite level are able to maintain physical performance output by MD -1 for a Saturday fixture.

8.3: Limitations of the thesis

Despite providing novel data, the research collected in this thesis is not without limitations, many of which were a direct consequence of working within an elite sport setting in an attempt to maintain ecological validity. The most prevalent limitation was missing data points. These occurred due to injuries and absences or limited testing time. Another limitation was not grouping players by position for the longitudinal analyses. Previous research has documented the differing demands of playing positions in elite footballers, with central and wide midfielders covering the greatest total distance and wide midfielders and full backs covering the most high-intensity running (Bangsbo, 1994; Bradley et al., 2009). If incorporated into this investigation, different effects of cumulative load on the conventional markers of recovery-fatigue may have been observed. However, where possible, position specific changes were incorporated using percentage change, ES change and/or graphical representations. Statistical significance testing was not included for position specific changes due to low sample sizes.

The greatest limitation, on reflection, was not undertaking our own reproducibility statistics. It cannot be assumed that the test-retest error would be comparable to previous literature. In order for practitioners to accurately distinguish between a 'true' meaningful change and test-retest error, it is vital that intra and inter-reliability are reported and compared to the changes observed. In addition, an unavoidable limitation when undertaking the research was a change in management/coaching that presents changes in coaching strategies and match starting formations, all factors that can affect physical output. Further, as previously discussed the observation of only one blood biomarker (CK) was a limitation to measuring fatigue when used alone due to the high intra and inter-individual variability in CK concentration. Lastly, the use of therapeutic interventions should be taken into consideration when interpreting the data. In elite

football, the high frequency of competition during the in-season phase ensures training is more focused around recovery and optimising physical fitness, which may lead to lesser changes in physiological and physical performance markers. In an ideal controlled scientific setting, therapeutic interventions used by the team to promote recovery would be removed (e.g soft tissue massage, ice, cold-water immersion, hot/cold therapy, compression garments and supplementation). However, in a professional sport setting, this was not possible. Nevertheless, what was lost in scientific rigour was more than compensated for in ecological validity.

8.4: Conclusions and recommendations for future research

The published research related to valid markers of fatigue in elite football is scarce and contradictory due to the inconsistency in study populations (e.g. level of playing standard and/or academy vs senior first team players), protocols (e.g. 'one-off' data collections and time-point of data collection) and equipment used (e.g. JJS vs. force platform and capillary blood vs. venous bloody testing). Nevertheless, in the current thesis CK concentrations and CMJ outputs (FT, FT:CT, AP, jump height and TV) displayed acute sensitivity to fatigue induced changes 48-h post 90-min of competitive match-play in elite senior football players. This observation was in agreement with some of the previously conducted research, but the differences in varying methodologies in the literature make it difficult to compare between studies. However, in all instances, a valid marker of fatigue should be sensitive to variability in preceding match/training loads to determine the effects of exercise duration and intensity on player's physiological and physical response (Meeusen et al., 2013). Yet, the longitudinal analyses of these recovery-fatigue markers revealed that some of these conventional markers of fatigue displayed fluctuations to baseline measures across the season but there was minimal association to training load outputs measured via GPS. The research findings that

comprise this thesis observed minimal sensitivity in these measures to preceding match/training load and a definitive conclusion regarding this could not be stated. Furthermore, it should be noted that training and match load was represented by distances covered or time spent training/playing competitive football. It could be argued that the low proportion of variance explained from GPS metrics was due to other factors not included in the analyses such as technical actions of passes, tackles, shots, headers, crossing and jumping to win an aerial ball (Bradley and Ade, 2018). Such high-force actions may contribute to non-metabolic fatigue of the muscular and physiological system, which can impair muscle function and subsequent performance (Hubal et al., 2007; Hedayatpour et al., 2009). The integration of these factors and their associations to recovery-fatigue variables would allow a more holistic insight into the myriad of factors that may contribute to fatigue in football.

In agreement with the findings by Nowakowska et al. (2019) it was concluded that although CK demonstrates large inter-individual variability it displays a weak positive association to cumulative match-time over the season, potentially being able to detect signs of cumulative fatigue if compared to individual baseline measures. However, in order to gain full understanding of a players' physiological response to training/game demands, future research should incorporate more detailed blood analysis. As microscopic muscle damage, inflammation and the associated acute response come successively during/after exercise, the observation of IL-6 may enhance decisions on muscular stress response to exercise. IL-6 is a cytokine that can indicate inflammation and is produced in larger amounts than any other cytokine, yet like many biomarkers still presents the same limitations of high inter-individual variability and is of little diagnostic value when used alone. Further, observing for chronically elevated levels of cortisol, a catabolic hormone, may indicate reduced ability for muscle adaptation and recovery from

match/training load. These biomarkers, when used in combination with other measurement tools such as CMJ performance may provide practitioners with valuable insights regarding the muscle damage, inflammatory response and recovery ability in elite level football players. However, appropriate reference ranges should be determined for specifics sub-groups and repeat measurements to allow practitioners to establish individual normative values and thus help the interpretation of meaningful changes from baseline measures.

Previous research has questioned the sensitivity of concentric focused outputs to detect signs of NF, yet the research that comprises this thesis suggest that CMJ height and FT are able to detect alteration in performance both acutely 48-h post 90-min of competitive match-play and longitudinally. Nevertheless, it appears that TV, AP and FT:CT may be more sensitive to cumulative fatigue induced changes and give an indication of NF in elite players. It is possible that these performance changes are caused by reductions in the amplitude of the counter-movement sub-consciously chosen by the athletes. Hence, quantifying changes in these kinematic variables may be of great interest for practitioners monitoring the fatiguing effects of training and competition over time. Elite level football players exhibit the capability to increase and/or maintain force outputs despite signs of low-level NF. These findings are in agreement with previous researching reporting that elite athletes are able to maintain performance through modification of movement strategies and/or skill (Knicker et al., 2011). Further, this cohort of elite senior level football players displays the ability to be adequately recovered by MD -1 and maintain physical performance despite the imposition of a mid-week fixture. Therefore, the structured daily undulating micro-cycles utilised in football are effective in reducing and evading the effect of fatigues by match-day for a scheduled Saturday fixture (Kelly et al., 2020).

Lastly, elite level footballers are able to maintain state of euhydration across the season. The time-point 48-h post-match allows sufficient time for players to rehydrate. However, the validity of Uosm testing has been questioned and future research should employ equipment that gives a more accurate measure of hydration status and not fluid turn-over (Cheuvront and Sawka, 2005).

It can be concluded that the aims and objectives set out in Chapter 1 of this thesis have been achieved. The research studies that comprise this thesis have provided practitioners with the knowledge to determine the effectiveness and sensitivity of the conventional measures of recovery-fatigue used in professional football and its applicability to elite senior football players. Considering the multifactorial nature of fatigue, further research questions generated from these findings include: 1) How might the integration of technical and tactical factors on physical performance associate to changes in recovery-fatigue variables? 2) How might the these minor alterations in physiological and physical performance impact subsequent physical and technical performance during matches and training? 2) Do changes in NF relate more closely to non-contact muscle/ligament injuries over that of physical and technical performance changes?

Finally, with the availability to now use GPS tracking software in all competitive league games, a more accurate indication of preceding/cumulative load can be analysed in relation to changes in markers of fatigue. Future research could investigate these match and training demands on markers of fatigue in elite individuals. In general, markers used need to be easy to implement, time efficient, easy to analyse and have good reliability to detect meaningful changes in performance. Jump testing is appealing due to its efficiency, non-fatiguing nature and readily available outputs that can provide valuable

insight into an athletes NF and SSC capability. Creatine kinase testing via capillary blood is also easy to administer and may provide insight into signs of cumulative fatigue over a season if interpreted correctly. This thesis has provided an understanding into the effect of match and training demands on acute and longitudinal changes in physiological and physical performance markers and their application to elite, senior, football players. The application of the data in this report is partly dependent on the extent that the information can be generalised to other populations of elite football players.

In summary, this thesis established that simple output measures from a CMJ test are able to detect impairments in physical performance and signs of NF at 48-h post-match. These concentric derived output measures from the push-off phase, previously criticised in research, showed sensitivity to match-external load completed during 90-mins of elite competitive football. Over a competitive season, elite players are able to maintain and/or increase MF and PP outputs from the CMJ despite alterations in how they may produce their force/power. The collection of plasma CK displayed sensitivity both acutely pre to post-match and cumulatively to match-min across the season. Therefore, in a real-world applied environment of elite senior level football players, the monitoring of simple output measures from a CMJ and for spikes in CK concentrations, in comparison to individual baseline measures, may provide clinically relevant insights into the recovery-fatigue process of individual players.

8.5: Practical application of fatigue monitoring in elite senior players

Based on the findings from the research presented in this thesis, the following practical recommendations are made for practitioners engaging in monitoring fatigue/recovery in elite football players via assessment and analysis of CMJ performance, CK concentration and Uosm.

CMJ performance:

- CMJ height measured via JJS is able to detect small changes in physical performance 48-h post competitive match play.
- Where possible a force platform/ force plate should be used to assess CMJ due to its greater precision and accuracy.
- CMJ FT, AP, FT:CT and TV are able to detect signs of NF 48-h post competitive match-play.
- CMJ AP and FT display the greatest sensitivity to match external load 48-h post-match.
- CMJ TV shows greater sensitivity to cumulative fatigue across a football season.
- Players are able to increase/maintain max force produced via a CMJ across a season.
- Players are able to maintain max force and peak power outputs despite signs of low-level NF via changes in 'jump-strategy' (e.g TV, FT:CT, AP).
- Physical performance from a CMJ is recovered by MD -1 for a scheduled Saturday fixture despite the imposition of mid-week fixture.

CK concentration:

- CK is sensitive to the overall demands of match-play but not highly associated with match external-load.
- CK displayed sensitivity to cumulative match-load across a season.
- The high typical variation in CK concentration between and within football players demonstrates the importance of determining thresholds for unusual changes using individual data.
- Observing changes from normative values via Z-scores can enhance interpretation of meaningful changes.

- Changes in CK concentrations should always be compared to the references ranges for that particular sub-group to understand the clinical significance of changes in raw data.
- The 95 % reference range for CK concentration in elite senior level football players across a competitive season is 98 to 1417 u/l.
- Additional biomarkers of exercise stress should be used in combination with CK to provide a greater representation of a player's physiological response to training/game demands.
- Practitioners should consider the time-point of data collection for the most appropriate biomarkers to observe as some have been shown to be normalised 24-h after a match.

Uosm:

- Provided that players have an appropriate hydration strategy in place, competitive football does not induce dehydration 48-h post-match in elite football players.
- Hydration strategies during a season allow players to maintain a 'euhydrated' state where performance is unlikely to be adversely affected.
- The 95 % reference range for Uosm in elite senior level football players across a competitive season is 137 to 675 mOsmols.

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Appendix A: Case Study of trends between injury incidence and CMJ, CK and Uosm.

Introduction

The studies that comprise this thesis have demonstrated that certain CMJ output measures of physical performance and plasma CK concentrations are sensitive to acute and chronic fluctuations in match/training load. Therefore, the observation of these markers can help practitioners determine the readiness of individual players, and aid to design and adjust individual player's recovery strategies to ensure peak performance on match-day (if analysed and interpreted correctly).

As discussed within the thesis, fixture congestion in elite football is of contemporary concern, requiring players to compete in up to three games per-week with as little as 72-h in-between matches (Barnes et al., 2014; Carling et al., 2015; Abaïda and Dupont, 2018). Previous research has demonstrated an increased injury risk in league matches with four or less days' recovery when compared with six or more days' recovery (Ekstrand et al., 2013). Injuries to key players and/or regular starters can be detrimental to the success of the team as a whole (Hägglund et al., 2013). Further, the link between training/match load and injury incidence has been extensively investigated in elite athletes suggesting that the harder the individual trains, the higher the likelihood of injury and illness (Gabbett and Ullah, 2012; Hulin et al., 2016). Thus, if a marker of fatigue is-sensitive to match/training load they may be able to detect when a player is at a higher risk of sustaining an injury. A possible link between these factors would allow practitioners to make adjustments, if needed, to the training regimen in order to avoid overtraining, and the occurrence of illness and injury could be reduced (Drew and Finch, 2016).

This case study was undertaken to observe any possible trends between non-contact injuries and changes in CMJ, CK and Uosm prior to injury incidence.

Injury Cases

The injuries included are those of a non-contact nature (muscle, ligament and cartilage). For the purpose of this investigation, injury data were only included where players had undergone recovery-fatigue testing the day prior to injury incidence occurring and had undertaken baseline testing at the start of the season for comparisons to be made. Table 1 shows the non-contact injuries sustained during the 2015-16 season that met the requirement for analysis.

Table 1. Injury overview of players who met the requirement for analysis.

Player ID	Mechanism	Affected Region	Injury Type	Month Injured	Days Missed
1	Running	Hamstring	Muscle	August	20
2	Sprinting	Hamstring	Muscle	September	19
3	Sprinting	abdomen	Muscle	January	5
4	Sprinting	Hamstring	Tendon	January	53
5	Sprinting	thigh	Muscle	February	26
6	twisting	knee	Cartilage	February	47
7	stretching	thigh	Muscle	March	5
8	Kicking	hip & Groin	Muscle	October	19

Statistical analysis

The CMJ, CK and Uosm data were analysed by change to individual training baseline (BAS) measures from the start of the competitive season (as per analyses performed in Chapter 6). In addition to this, to replicate the analyses performed in Chapter 7 of this thesis, data were analysed by individual Z-score changes from the absolute season average. A Z-score represents the number of standard deviations that the response is above or below the mean of the distribution.

Table 2 shows the normative % change observed in each recovery-fatigue measure as detailed in Chapter 5 and 6 of the thesis, and the CV % as reported in the literature for elite athletes (Chapter 2.8.3; Table 2.4). The inclusion of this data was to aid the interpretation of any possible deviations outside of what is deemed 'normal' for elite football players in response to training and match-play. Graphs were included for data that showed a deviation outside the 'norm' as indicated from Table 2.

Table 2. Normative percentage (%) change in CMJ, CK and Uosm as described in Chapter 5 and 6 of this thesis, and CV % reported from previous research in elite athletes (Chapter 2.8.3).

	% fluctuation over a season (Chapter 6)	**Post-match % change (Chapter 5)**	**CV %**
Uosm	30 to 131*		34
CK	62 to 159*	49	18 to 20
CMJ height	-5.4 to -11.3*	4.2	3.8 to 5
CMJ CT	3.3*	2.2	
CMJ FT	-2.9 to -4.8*	-2.4	3.3
CMJ FT:CT	-7.5 to -12.4*	-7.4	
CMJ PP	-3.1 to 2.5	-4.5	2.9
CMJ MF	5.1 to 7*	3	2.2
CMJ TV	-3 to -6*	-3.3	2.5
CMJ AF	-2.5 to -2.8	0.2	1
CMJ AP	-9.4 to -11.5*	7.3	5

**Reported as statistically significant change to baseline values ($P < 0.05$)*

Results

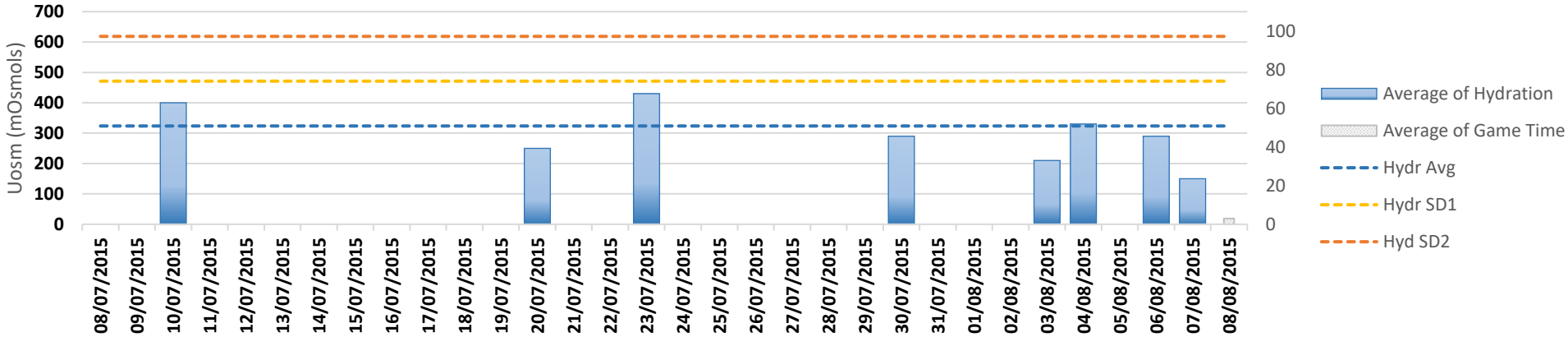
Table 3.a to 3.h displays each individual player's data prior to injury incidence. Player 7 showed the greatest deviation from baseline measures and Z-score change in CK and Uosm. Player 7 also showed significant deviations from baseline measures in CMJ height, CT, FT, FT:CT and TV (-4.8 to -12.9 %) prior to injury incidence. Similarly, player 4 demonstrated significant decrements in CMJ physical performance outputs CT, FT, FT:CT, AP and AF (-4.7 to -14.8 %) prior to injury incidence. Player 2 and player 6 displayed significant increases in CK prior to injury incidence in comparison to baseline measures (+73 to 113 %) and Z-score changes of 1.23 and 1.38, respectively. However, the clinical significance of this change for player 6 is questionable as pre-injury CK concentration of 376 u/l is relatively low when compared to the large reference range reported in Chapter 6 of this thesis. Nevertheless, player 2 also showed a Z-score change of 1.37 in CMJ CT and increases in comparison to baseline measures (+6.1 %) suggesting they were taking longer to produce peak outputs. Players 3 and 5 showed no significant changes in CK, Uosm, or CMJ physical performance prior to injury incidence. Lastly, there were four players (3, 5, 6 and 8) that showed improvements in CMJ physical performance output prior to injury incidence.

Player 1

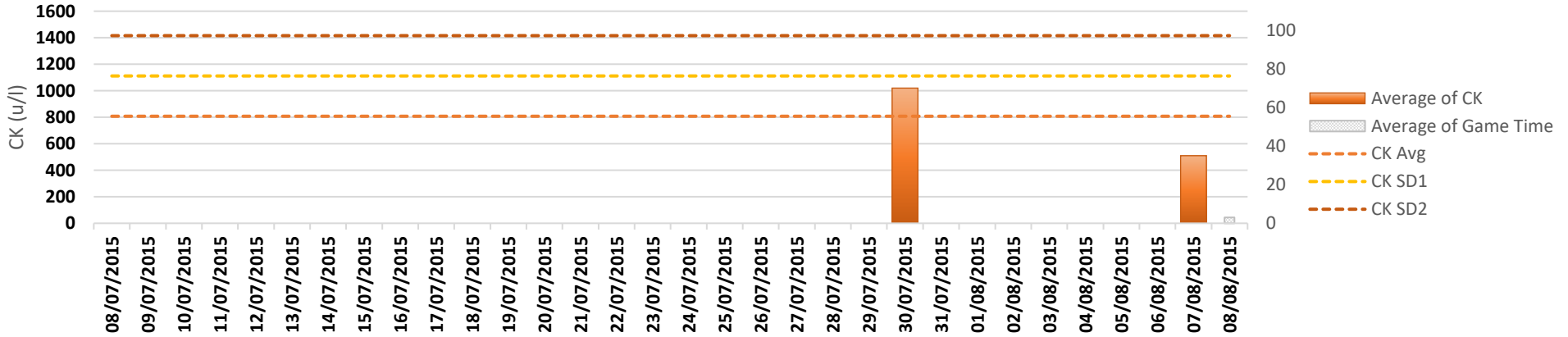
Table 1.a. Player 1 pre-injury data comparison to baseline measures and absolute average.

	Uosm	CMJ	CT	FT	FT:CT	PP	MF	TV	AP	AF	CK
Pre-injury testing	150	40.4	870.8	601.7	0.69	5142.2	2099.05	2.82	1330.8	1226.9	510
Training BAS	290	40.3	903.3	595.8	0.66	5130.1	2068.54	2.81	1325.6	1222.1	1020
Change from BAS	-140	0.1	-32.5	5.8	0.03	12.1	30.5	0.01	5.2	4.8	-510
% from BAS	-48 %	0.4 %	-3.6 %	1.0 %	4.7 %	0.2 %	1.5 %	0.36 %	0.4 %	0.39%	-50 %
Z-score change	-1.12	-0.35	-0.29	-0.03	0.13	-1.92	-1.45	-0.30	-0.02	-0.92	-1.03

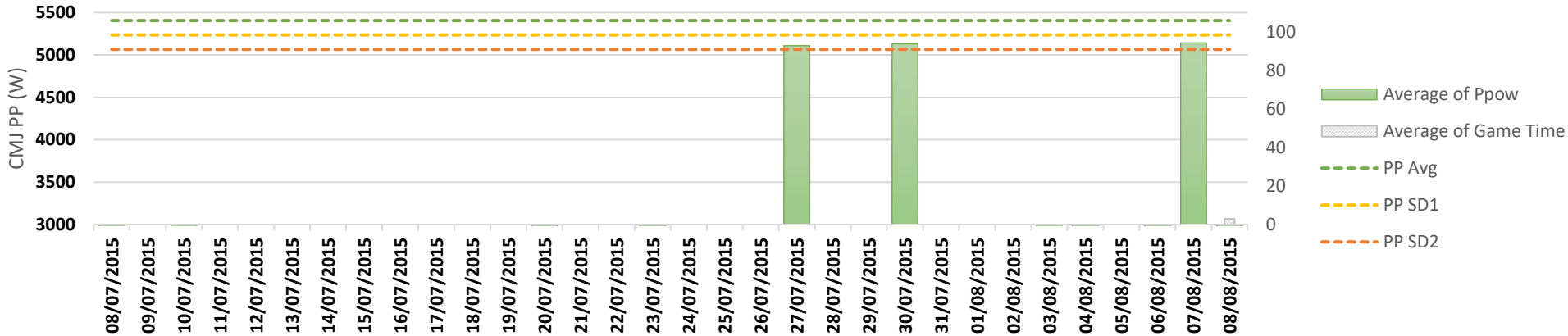
Hydration



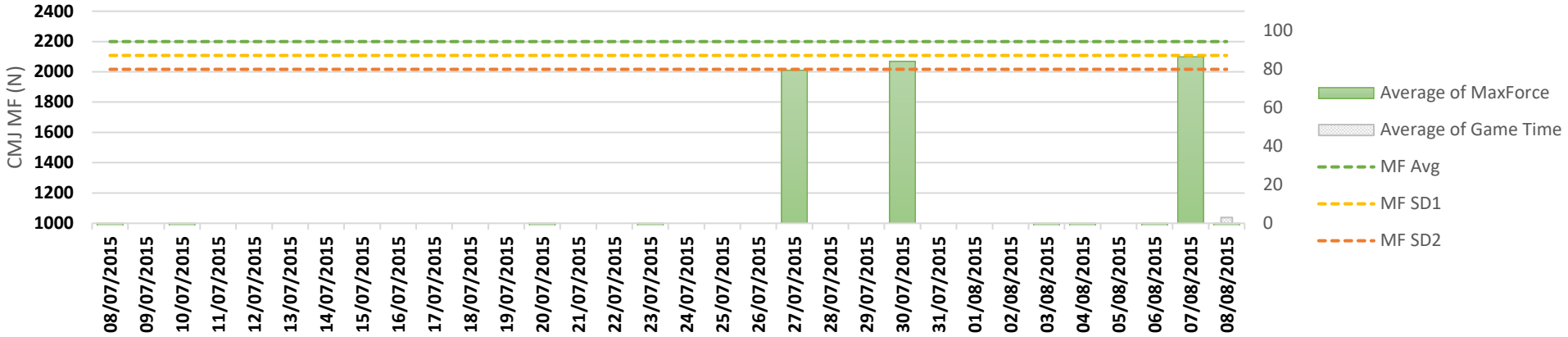
CK


1600
1400
1200
1000
800
600
400
200
0
CK (u/l)
100
80
60
40
20
0
08/07/2015
09/07/2015
10/07/2015
11/07/2015
12/07/2015
13/07/2015
14/07/2015
15/07/2015
16/07/2015
17/07/2015
18/07/2015
19/07/2015
20/07/2015
21/07/2015
22/07/2015
23/07/2015
24/07/2015
25/07/2015
26/07/2015
27/07/2015
28/07/2015
29/07/2015
30/07/2015
31/07/2015
01/08/2015
02/08/2015
03/08/2015
04/08/2015
05/08/2015
06/08/2015
07/08/2015
08/08/2015
Average of CK
Average of Game Time
CK Avg
CK SD1
CK SD2

CMJ PP


5500
5000
4500
4000
3500
3000
CMJ PP (W)
100
80
60
40
20
0
08/07/2015
09/07/2015
10/07/2015
11/07/2015
12/07/2015
13/07/2015
14/07/2015
15/07/2015
16/07/2015
17/07/2015
18/07/2015
19/07/2015
20/07/2015
21/07/2015
22/07/2015
23/07/2015
24/07/2015
25/07/2015
26/07/2015
27/07/2015
28/07/2015
29/07/2015
30/07/2015
31/07/2015
01/08/2015
02/08/2015
03/08/2015
04/08/2015
05/08/2015
06/08/2015
07/08/2015
08/08/2015
Average of Ppow
Average of Game Time
PP Avg
PP SD1
PP SD2

CMJ MF

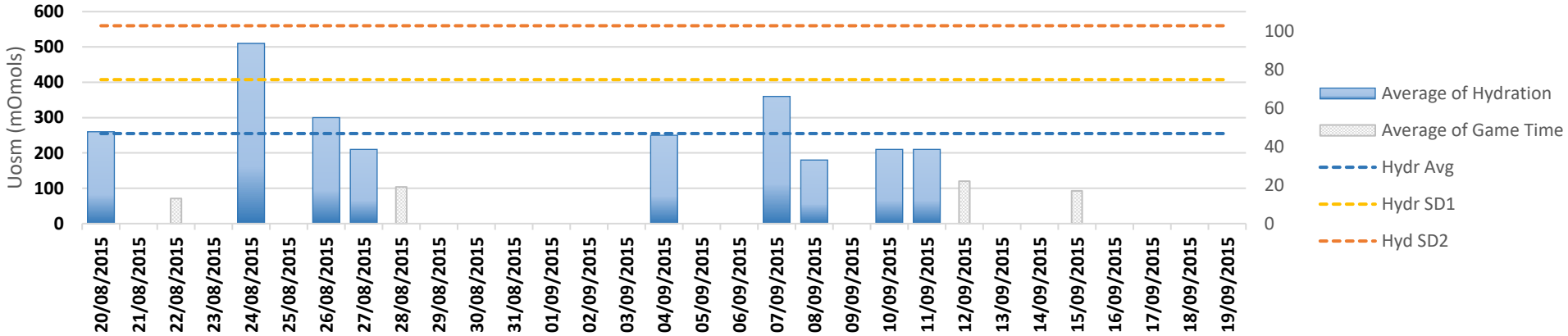

CMJ MF (N)
2400
2200
2000
1800
1600
1400
1200
1000
100
80
60
40
20
0
08/07/2015
09/07/2015
10/07/2015
11/07/2015
12/07/2015
13/07/2015
14/07/2015
15/07/2015
16/07/2015
17/07/2015
18/07/2015
19/07/2015
20/07/2015
21/07/2015
22/07/2015
23/07/2015
24/07/2015
25/07/2015
26/07/2015
27/07/2015
28/07/2015
29/07/2015
30/07/2015
31/07/2015
01/08/2015
02/08/2015
03/08/2015
04/08/2015
05/08/2015
06/08/2015
07/08/2015
08/08/2015
Average of MaxForce
Average of Game Time
MF Avg
MF SD1
MF SD2

Player 2

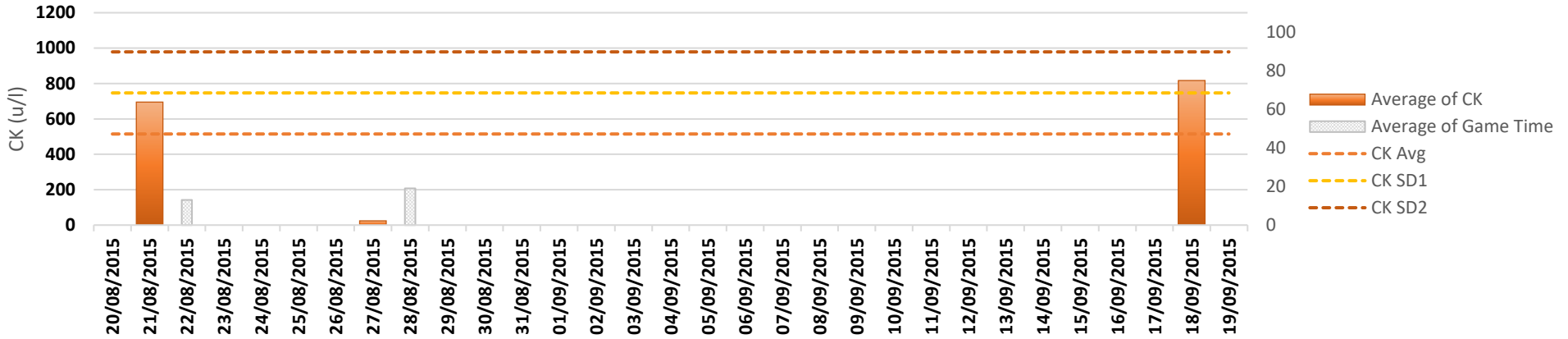
Table 1.b. Player 2 pre-injury data comparison to baseline measures and absolute average.

	Uosm	CMJ	CT	FT	FT:CT	PP	MF	TV	AP	AF	CK
Pre-injury testing		34.9	796.7	538.3	0.68	4794.2	2168.1	2.61	1243.2	1231.6	817
Training BAS		35.7	750.8	545.8	0.73	4810.3	2225.2	2.64	1294.4	1180.8	383
Change from BAS		-0.8	44.8	-7.5	0.05	-16.1	-57.2	0.03	-51.2	50.8	434
% from BAS		-2.3 %	6.1 %	-1.4 %	-7.0 %	-0.3 %	-2.6 %	-1.1 %	-4.0 %	4.3 %	113.3 %
Z-score change		-0.17	1.37	-0.43	-0.16	-0.28	-0.47	-0.16	-0.49	-0.39	1.23

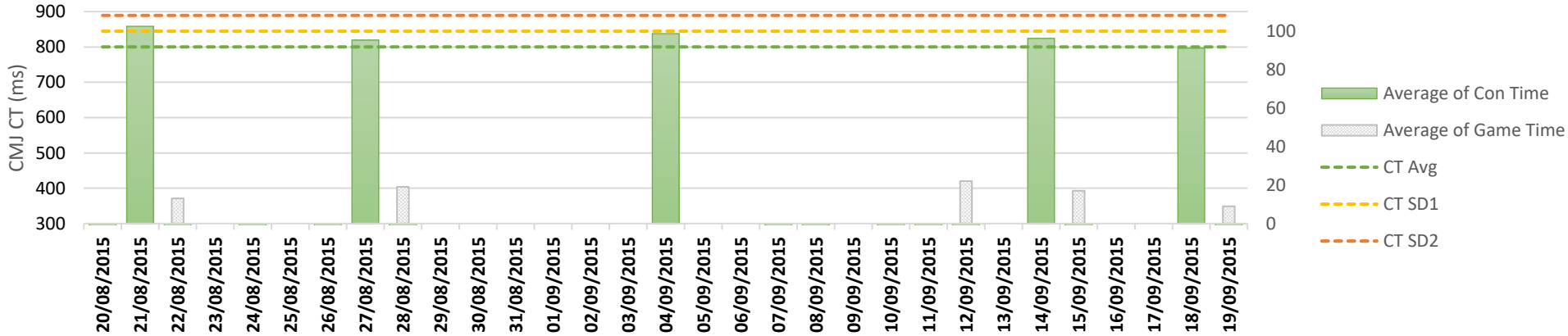
Hydration



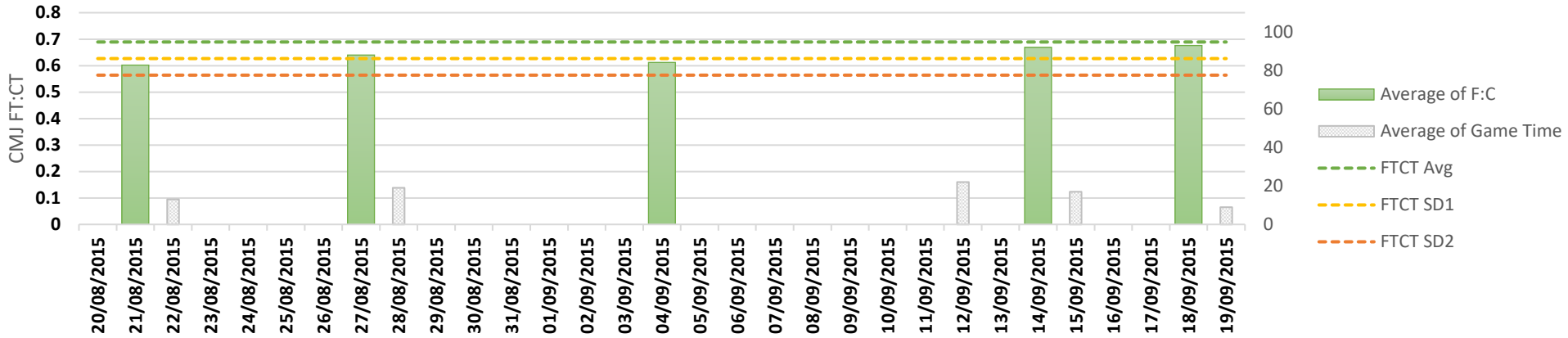
CK


CK (u/l)
1200
1000
800
600
400
200
0
100
80
60
40
20
0
20/08/2015
21/08/2015
22/08/2015
23/08/2015
24/08/2015
25/08/2015
26/08/2015
27/08/2015
28/08/2015
29/08/2015
30/08/2015
31/08/2015
01/09/2015
02/09/2015
03/09/2015
04/09/2015
05/09/2015
06/09/2015
07/09/2015
08/09/2015
09/09/2015
10/09/2015
11/09/2015
12/09/2015
13/09/2015
14/09/2015
15/09/2015
16/09/2015
17/09/2015
18/09/2015
19/09/2015
Average of CK
Average of Game Time
CK Avg
CK SD1
CK SD2

CMJ CT


CMJ CT (ms)
900
800
700
600
500
400
300
100
80
60
40
20
0
20/08/2015
21/08/2015
22/08/2015
23/08/2015
24/08/2015
25/08/2015
26/08/2015
27/08/2015
28/08/2015
29/08/2015
30/08/2015
31/08/2015
01/09/2015
02/09/2015
03/09/2015
04/09/2015
05/09/2015
06/09/2015
07/09/2015
08/09/2015
09/09/2015
10/09/2015
11/09/2015
12/09/2015
13/09/2015
14/09/2015
15/09/2015
16/09/2015
17/09/2015
18/09/2015
19/09/2015
Average of Con Time
Average of Game Time
CT Avg
CT SD1
CT SD2

CMJ FT:CT


CMJ FT:CT
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
100
80
60
40
20
0
20/08/2015
21/08/2015
22/08/2015
23/08/2015
24/08/2015
25/08/2015
26/08/2015
27/08/2015
28/08/2015
29/08/2015
30/08/2015
31/08/2015
01/09/2015
02/09/2015
03/09/2015
04/09/2015
05/09/2015
06/09/2015
07/09/2015
08/09/2015
09/09/2015
10/09/2015
11/09/2015
12/09/2015
13/09/2015
14/09/2015
15/09/2015
16/09/2015
17/09/2015
18/09/2015
19/09/2015
Average of F:C
Average of Game Time
FTCT Avg
FTCT SD1
FTCT SD2

Player 3

Table 1.c. Player 3 pre-injury data comparison to baseline measures and absolute average.

	Uosm	CMJ JH	CT	FT	FT:CT	PP	MF	TV	AP	AF	CK
Pre-injury testing	330	31.98	785.0	545.8	0.695	4047.0	1914.2	2.51	923.2	1054.1	
Training BAS	360	33.32	895.0	554.2	0.619	3909.1	1828.7	2.56	888.0	1015.6	
Change from BAS	-30	-1.3	-110.0	-8.3	0.08	138.0	85.6	-0.05	35.2	38.6	
% from BAS	-8.3 %	-4.0 %	-12.3 %	-1.5 %	12.3 %	3.5 %	4.7 %	-2.0 %	4.0 %	3.8 %	
Z-score change	0.64	-0.75	-0.67	-0.56	0.12	-0.22	0.39	-0.66	-0.99	0.11	

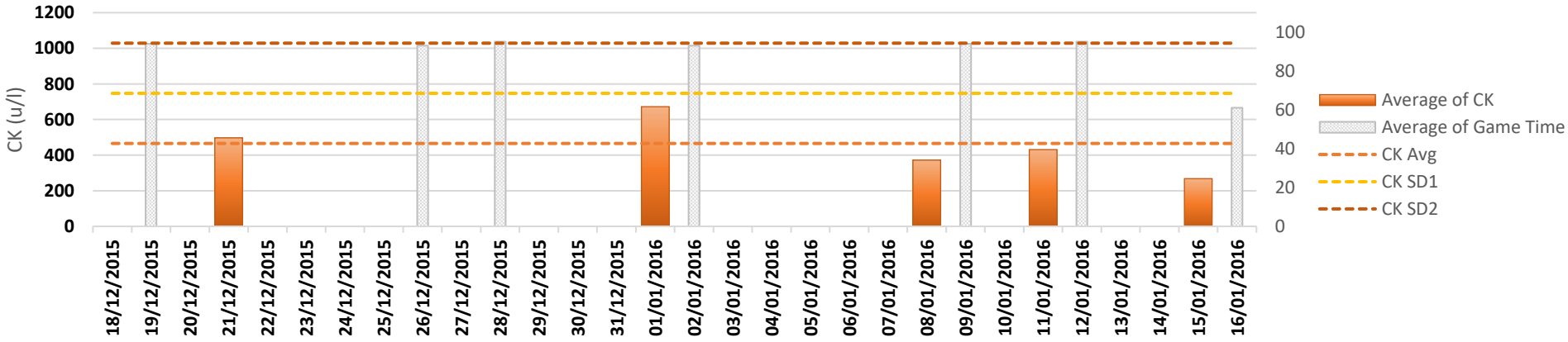
Player 4

Table 1.d. Player 4 pre-injury data comparison to baseline measures and absolute average.

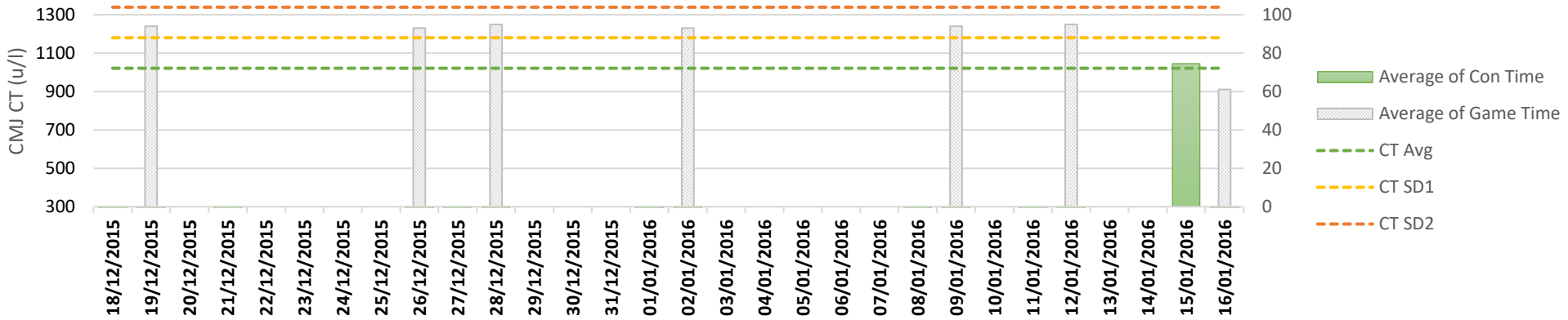
	Uosm	CMJ JH	CT	FT	FT:CT	PP	MF	TV	AP	AF	CK
Pre-injury testing		38.7	1045.0	581.7	0.56	5144.9	2047.1	2.76	1142.0	1184.3	268
Training BAS		39.3	961.7	606.7	0.63	5228.2	2072.9	2.78	1340.7	1243.1	183
Change from BAS		-0.6	83.3	-25.0	-0.07	-83.3	-25.9	-0.02	-198.6	-58.8	85
% from BAS		-1.6 %	8.7 %	-4.1 %	-11.7 %	-1.6 %	-1.3 %	-0.7 %	-14.8 %	-4.7 %	46.5 %
Z-score change		-0.37	0.11	-0.29	-0.42	-0.38	-0.39	-0.24	-0.65	-0.41	0.09

Player 4:

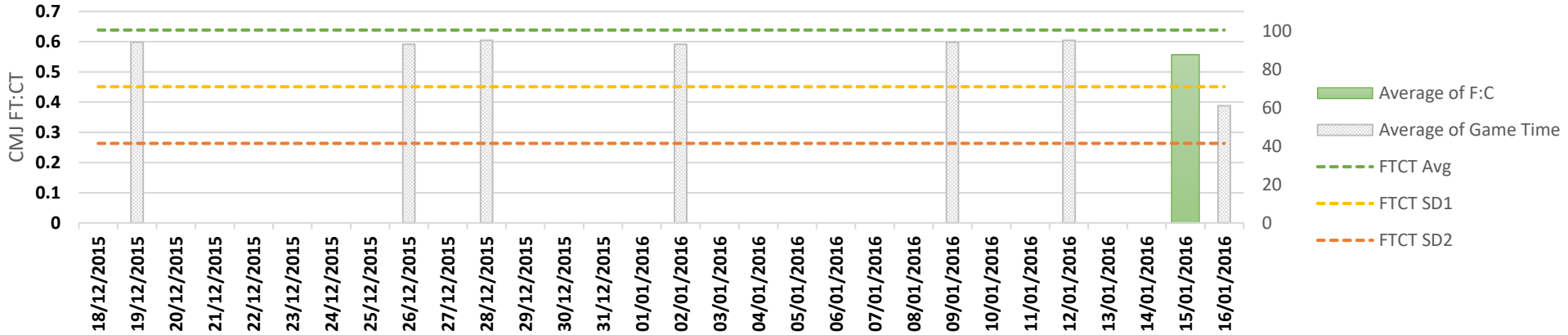
CK



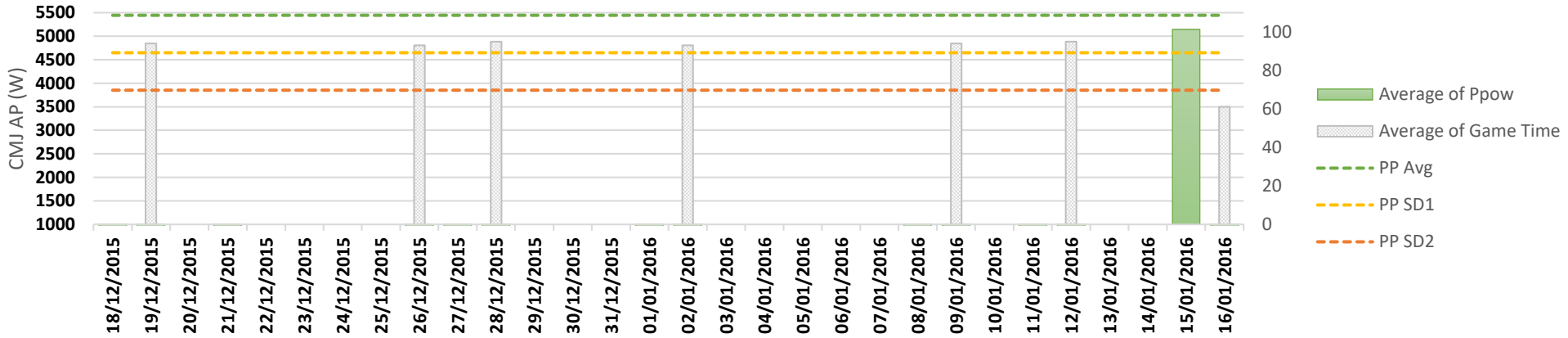
CMJ CT



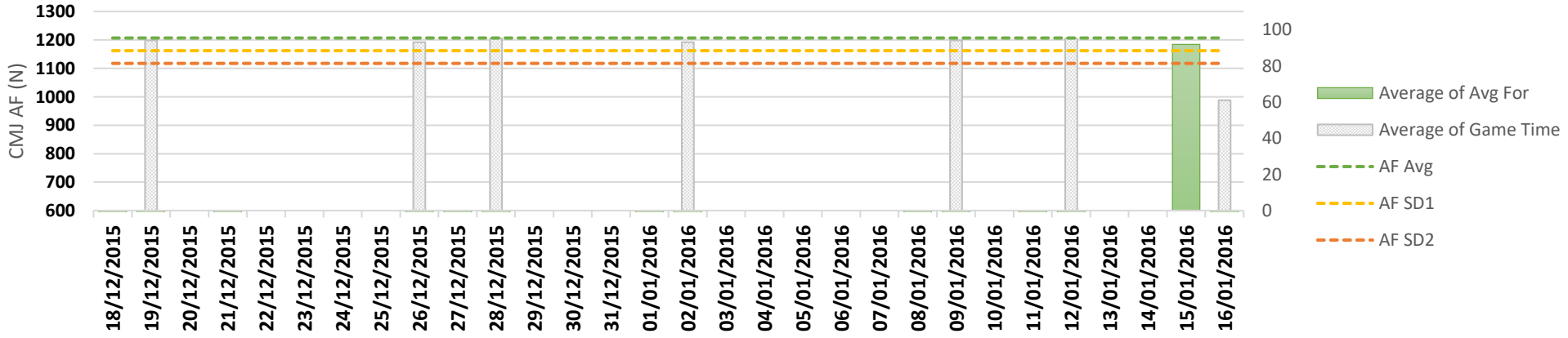
CMJ FT:CT



CMJ AP


CMJ AP (W)
5500
5000
4500
4000
3500
3000
2500
2000
1500
1000
100
80
60
40
20
0
18/12/2015
19/12/2015
20/12/2015
21/12/2015
22/12/2015
23/12/2015
24/12/2015
25/12/2015
26/12/2015
27/12/2015
28/12/2015
29/12/2015
30/12/2015
31/12/2015
01/01/2016
02/01/2016
03/01/2016
04/01/2016
05/01/2016
06/01/2016
07/01/2016
08/01/2016
09/01/2016
10/01/2016
11/01/2016
12/01/2016
13/01/2016
14/01/2016
15/01/2016
16/01/2016
Average of Ppow
Average of Game Time
PP Avg
PP SD1
PP SD2

CMJ AF


CMJ AF (N)
1300
1200
1100
1000
900
800
700
600
100
80
60
40
20
0
18/12/2015
19/12/2015
20/12/2015
21/12/2015
22/12/2015
23/12/2015
24/12/2015
25/12/2015
26/12/2015
27/12/2015
28/12/2015
29/12/2015
30/12/2015
31/12/2015
01/01/2016
02/01/2016
03/01/2016
04/01/2016
05/01/2016
06/01/2016
07/01/2016
08/01/2016
09/01/2016
10/01/2016
11/01/2016
12/01/2016
13/01/2016
14/01/2016
15/01/2016
16/01/2016
Average of Avg For
Average of Game Time
AF Avg
AF SD1
AF SD2

Player 5

Table 1.e. Player 5 pre-injury data comparison to baseline measures and absolute average.

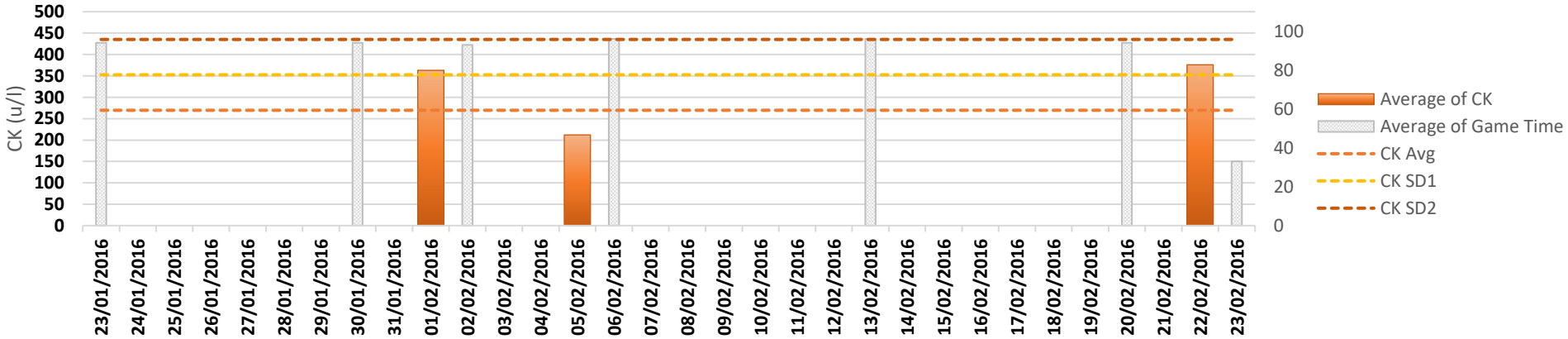
	Uosm	CMJ JH	CT	FT	FT:CT	PP	MF	TV	AP	AF	CK
Pre-injury testing		43.1	836.7	618.3	0.739	5515.5	2171.1	2.91	1427.7	1268.1	705
Training BAS		40.3	903.3	595.8	0.66	5130.1	2068.5	2.81	1325.6	1222.1	1020
Change from BAS		2.8	-66.7	22.5	0.08	385.4	102.6	0.10	102.1	46.0	-315
% from BAS		7.0 %	-7.4 %	3.8 %	12.0 %	7.5 %	5.0 %	3.6 %	7.7 %	3.8 %	-31 %
Z-score change		0.92	-0.97	0.56	0.94	0.55	-0.55	0.95	0.72	0.84	-0.39

Player 6

Table 1.f. Player 6 pre-injury data comparison to baseline measures and absolute average.

	Uosm	CMJ JH	CT	FT	FT:CT	PP	MF	TV	AP	AF	CK
Pre-injury testing	180	36.9	814.2	570.8	0.70	4672.4	2103.4	2.69	1294.5	1199.06	376
Training BAS	450	37.2	858.3	580.0	0.68	4457.8	2016.6	2.64	1294.4	1180.77	218
Change from BAS	-270	-0.3	-44.2	-9.2	0.02	214.6	86.8	0.05	0.1	18.3	158
% from BAS	-60 %	-0.8 %	-5.2 %	-1.6 %	3.7 %	4.81%	4.3 %	1.9 %	0.01 %	1.6 %	73 %
Z-score change	-1.40	1.00	-0.37	0.83	0.57	1.49	0.82	0.98	0.42	1.02	1.38

CK

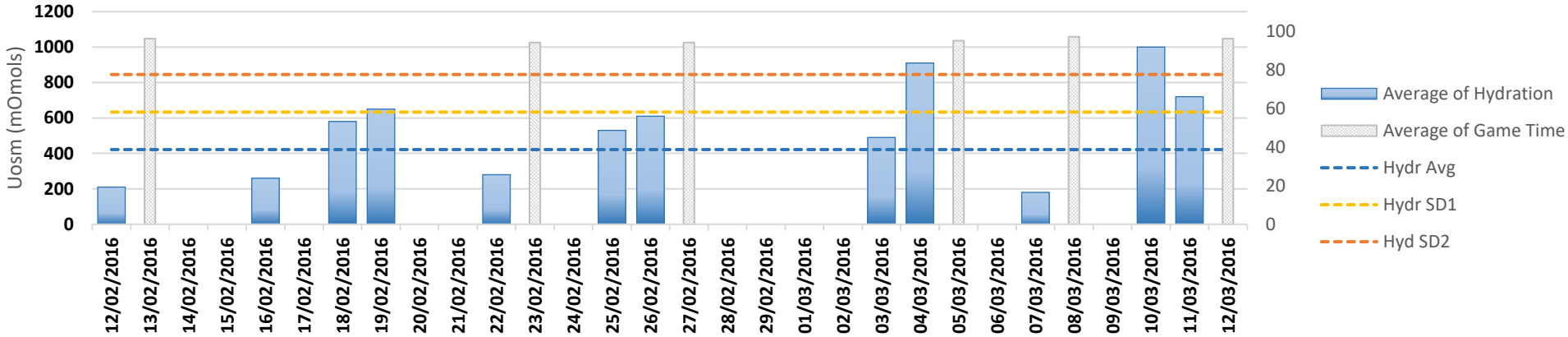


Player 7

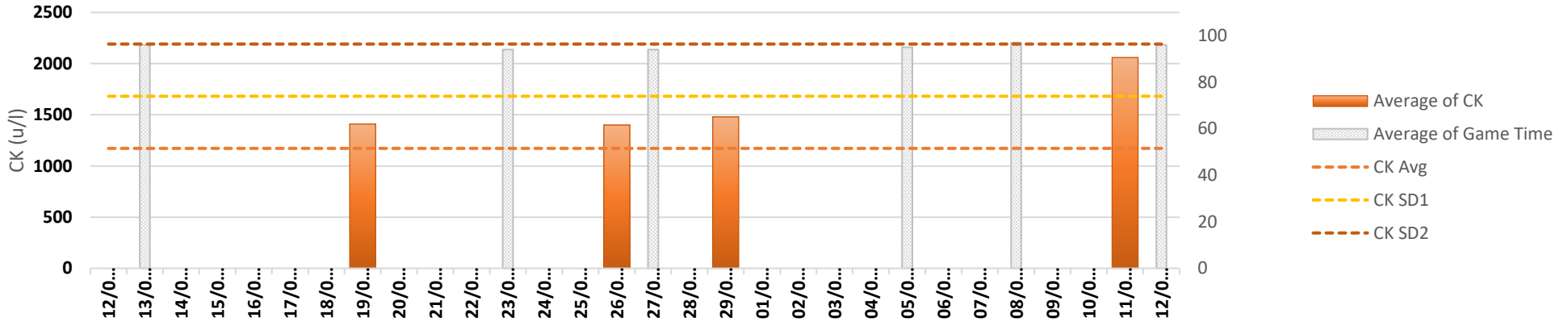
Table 1.g. Player 7 pre-injury data comparison to baseline measures and absolute average.

	Uosm	CMJ JH	CT	FT	FT:CT	PP	MF	TV	AP	AF	CK
Pre-injury testing	720	39.3	945	562.5	0.60	4210.6	1692.3	2.78	991.6	983.6	2060
Training BAS	470	43.5	875	597.5	0.68	4131.7	1723.7	2.92	942.1	930.4	300
Change from BAS	250	-4.2	70	-35.0	-0.09	78.8	-31.4	-0.14	49.5	53.2	1760
% from BAS	53.2 %	-9.6 %	8.0 %	-5.9 %	-12.9 %	1.9 %	-1.8 %	-4.8 %	5.3 %	5.7 %	587 %
Z-score change	1.3	-0.2	0.3	-0.2	-0.5	0.5	-0.4	-0.1	-0.1	0.2	1.9

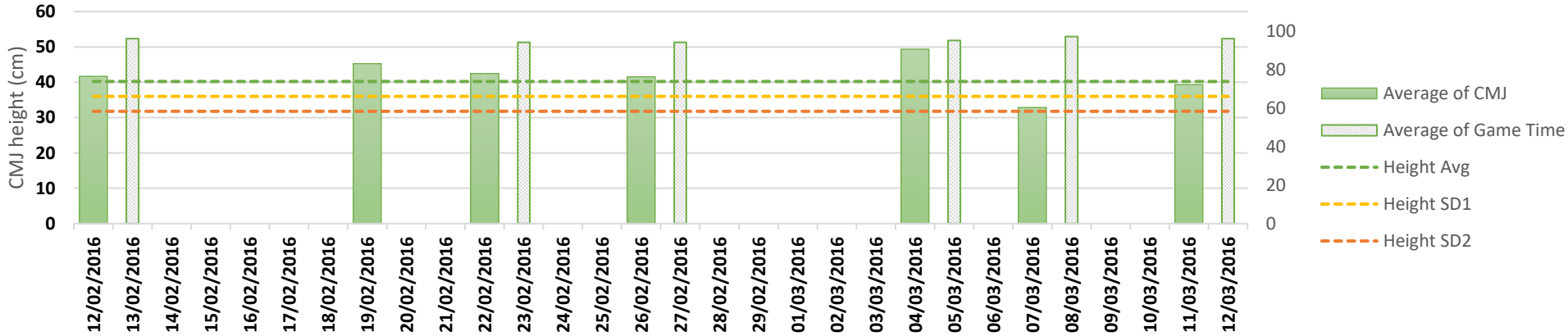
Hydration



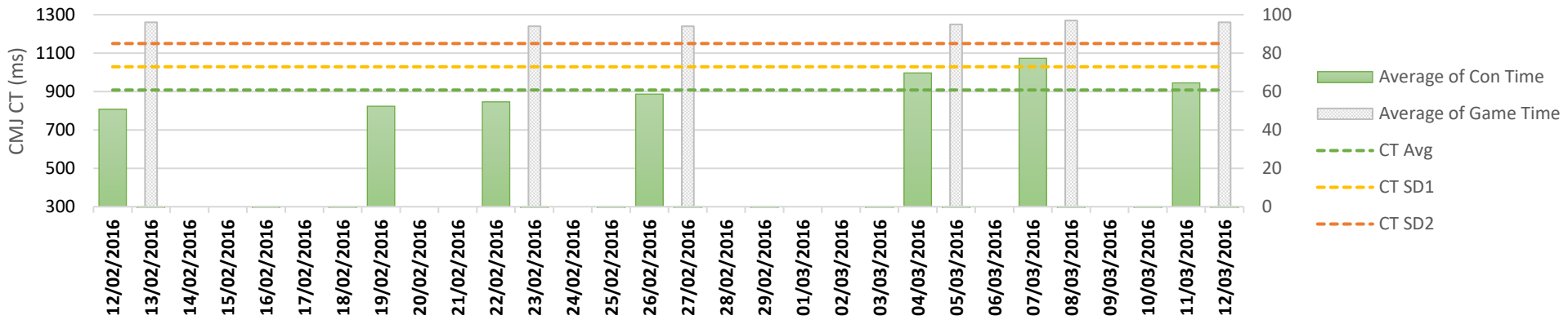
CK



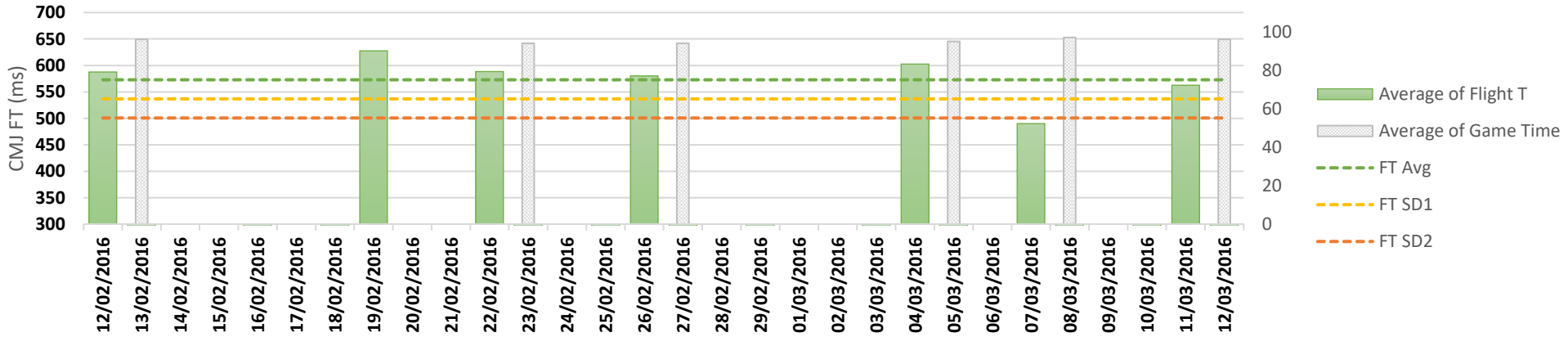
CMJ Height



CMJ CT


CMJ CT (ms)
1300
1100
900
700
500
300
100
80
60
40
20
0
12/02/2016
13/02/2016
14/02/2016
15/02/2016
16/02/2016
17/02/2016
18/02/2016
19/02/2016
20/02/2016
21/02/2016
22/02/2016
23/02/2016
24/02/2016
25/02/2016
26/02/2016
27/02/2016
28/02/2016
29/02/2016
01/03/2016
02/03/2016
03/03/2016
04/03/2016
05/03/2016
06/03/2016
07/03/2016
08/03/2016
09/03/2016
10/03/2016
11/03/2016
12/03/2016
Average of Con Time
Average of Game Time
CT Avg
CT SD1
CT SD2

CMJ FT


CMJ FT (ms)
700
650
600
550
500
450
400
350
300
100
80
60
40
20
0
12/02/2016
13/02/2016
14/02/2016
15/02/2016
16/02/2016
17/02/2016
18/02/2016
19/02/2016
20/02/2016
21/02/2016
22/02/2016
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26/02/2016
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01/03/2016
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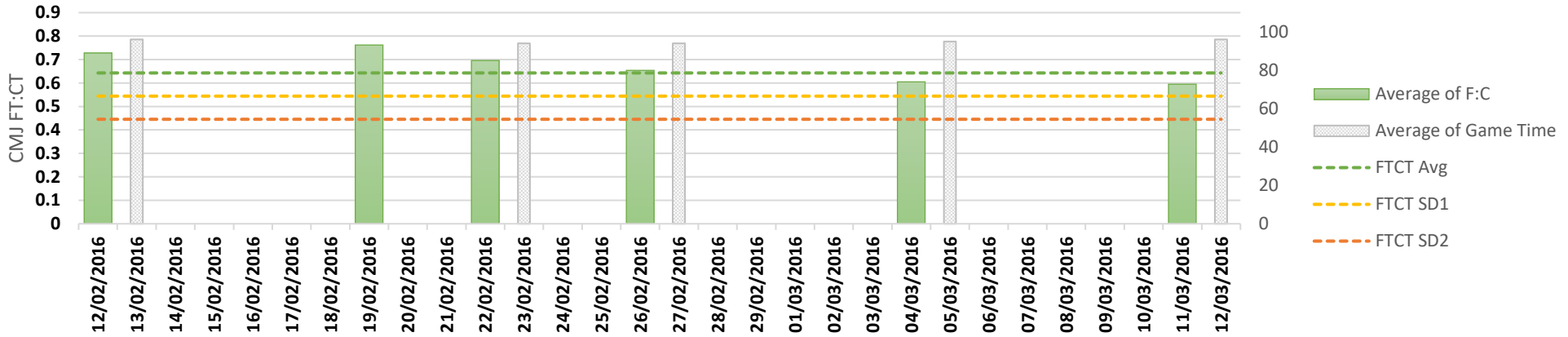
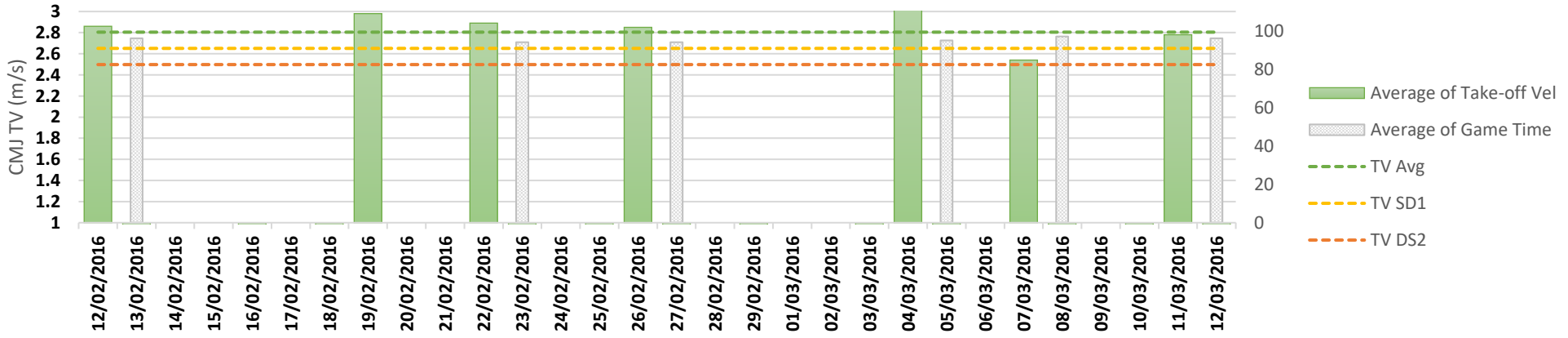
03/03/2016
04/03/2016
05/03/2016
06/03/2016
07/03/2016
08/03/2016
09/03/2016
10/03/2016
11/03/2016
12/03/2016
Average of Flight T
Average of Game Time
FT Avg
FT SD1
FT SD2

CMJ FT:CT



CMJ TV

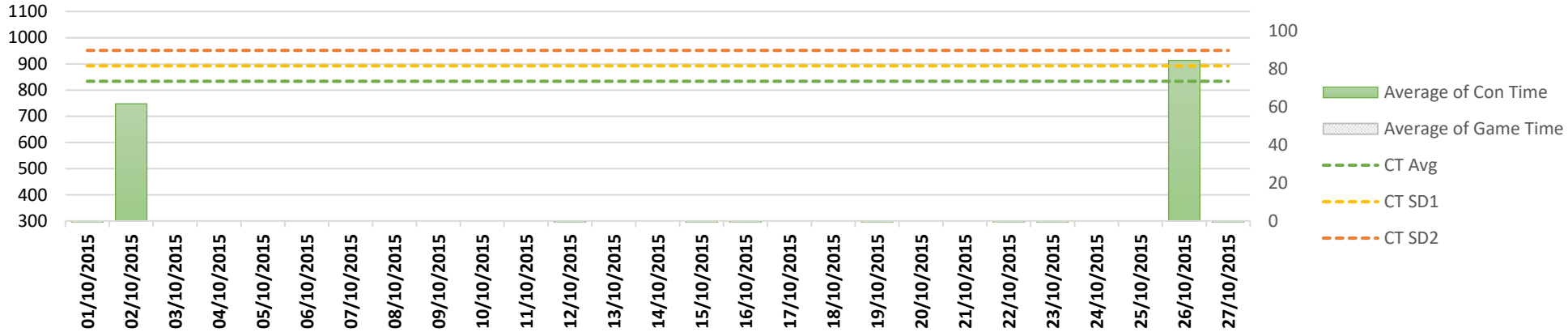


Player 8

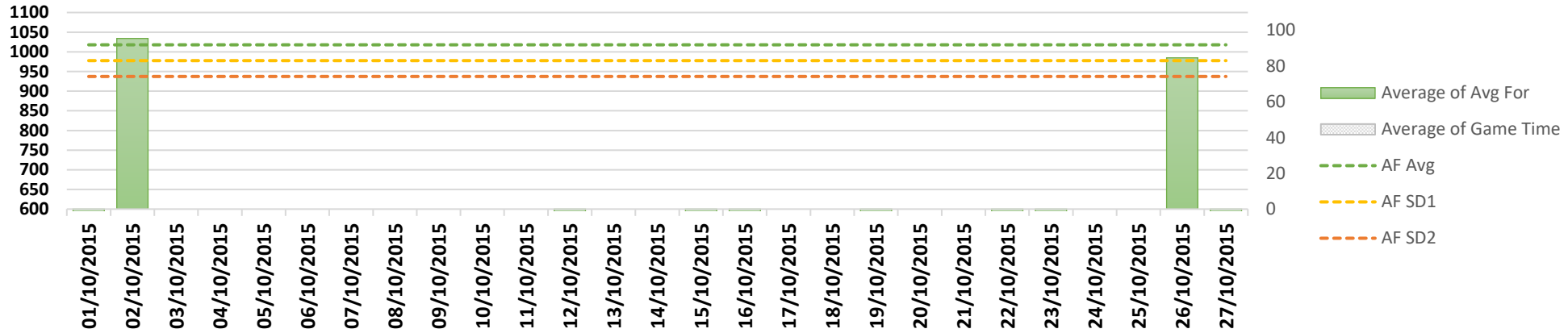
Table 1.h. Player 8 pre-injury data comparison to baseline measures and absolute average.

	Uosm	CMJ JH	CT	FT	FT:CT	PP	MF	TV	AP	AF	CK
Pre-injury testing	400	35.5	913.3	559.2	0.61	4138.2	1941.0	2.64	875.0	984.7	
Training BAS	330	31.4	958.3	530.0	0.55	3627.7	1707.2	2.48	819.7	960.2	
Change from BAS	70	4	-45.0	29.2	0.06	510.5	233.7	0.16	55.3	24.5	
% from BAS	21.2 %	12.9 %	-4.70%	5.5 %	10.7 %	14.1 %	13.7 %	6.5 %	6.7 %	2.6 %	
Z-score change	0.28	0.83	1.71	2.19	-0.93	1.48	0.81	0.86	-0.97	-1.04	

CMJ CT



CMJ AF


1100
1050
1000
950
900
850
800
750
700
650
600
100
80
60
40
20
0
01/10/2015
02/10/2015
03/10/2015
04/10/2015
05/10/2015
06/10/2015
07/10/2015
08/10/2015
09/10/2015
10/10/2015
11/10/2015
12/10/2015
13/10/2015
14/10/2015
15/10/2015
16/10/2015
17/10/2015
18/10/2015
19/10/2015
20/10/2015
21/10/2015
22/10/2015
23/10/2015
24/10/2015
25/10/2015
26/10/2015
27/10/2015
Average of Avg For
Average of Game Time
AF Avg
AF SD1
AF SD2

Conclusion

The data displayed no pattern or trend prior to injury incidence. There were observable decrements in CMJ physical performance for select players pre-injury but this was not consistent, with some players displaying improvements in performance. This inconsistency was also noted in CK concentrations and Uosm. Therefore, this sample suggests that these markers are not consistently sensitive to fatigue induced changes between individuals that may lead to injury, particularly of a soft-tissue nature. As previously stated in the thesis, the inclusion of additional biomarkers alongside CK may provide further insight into alterations in physiological status.

The occurrence of injury is as multifaceted and as complex as fatigue in team sports. There are many factors that may place a higher risk on players sustaining an injury (Jones et al., 2017), such as previous injury patterns and age of the athlete (Hagglund et al., 2006; Ekstrand et al., 2011). There is currently limited research available on objective fatigue markers and injury risk. However, the relationship between perceptual wellness scales and injury risk has been investigated more extensively (Jones et al., 2017). Large datasets are required over several seasons to accurately conclude on any possible links between fatigue markers and injury risk. Further, there are contrasting views on how to identify meaningful changes in response to training and match load beyond what is "normal" or random variability in the data (Carling et al., 2018). The standard deviation/ Z-score, effect size, smallest worthwhile change, coefficient of variation and percentage change are all examples of metrics used to determine this meaningful change (Robertson et al., 2017). As there is no standardised way and each metric will produce a different output, it makes investigating the relationship between these outputs and injury more challenging. Therefore, further research is required to determine whether these physiological and physical markers are linked to injury incidence.

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Appendix B: Published manuscript